American Journal of Diseases of Children

VOLUME 35 JUNE, 1928 NUMBER 6

THE ETIOLOGY OF AN EPIDEMIC OF ENTERITIS ASSOCIATED WITH MASTOIDITIS IN INFANTS *

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In 1927, there was a sudden increase in the death rate at The Cradle, an institution which cares for homeless infants from the time of birth till their adoption or disposal in appropriate institutions. The death rate, which in 1923, 1924, 1925 and 1926 had averaged 4.8 per cent, increased to 12.1 per cent in the late summer of 1927. This increase was due to an epidemic in which twenty-seven infants died and sixty-one more became ill but recovered, making a morbidity of 39.5 per cent in the institution. Among the infants who became ill during the epidemic, 30.7 per cent died.

The epidemic disease was apparently a clinical entity characterized by sudden onset with diarrhea and loss of weight. The stools were grayish yellow and watery without macroscopic blood or much increase in mucus. The loss of weight was progressive until recovery began or death occurred. Regurgitation was frequent. Sometimes at the onset, and as a rule later in the disease, there was an irregular fever, with a temperature varying between 97 and 100 to 104 F. rectally. A typical clinical chart shows the weight curve falling as the temperature rises.

In the infants who recovered, gradual improvement began from three days to two weeks after the onset of the disease. In fatal cases death occurred from the second to the nineteenth day of illness. In some fatal cases there was clinical evidence of a terminal bronchopneumonia, and in one instance there were symptoms of meningitis shortly before death.

The tympanic membranes were incised in eleven cases, and operations for drainage of the mastoid were done in five of the twenty-seven fatal cases. Surgical drainage of the mastoid cells did not affect the course of the disease as compared with the cases in which the operation was not done. All of the five on whom the operation was performed died.

* Received for publication, Jan. 20, 1928.

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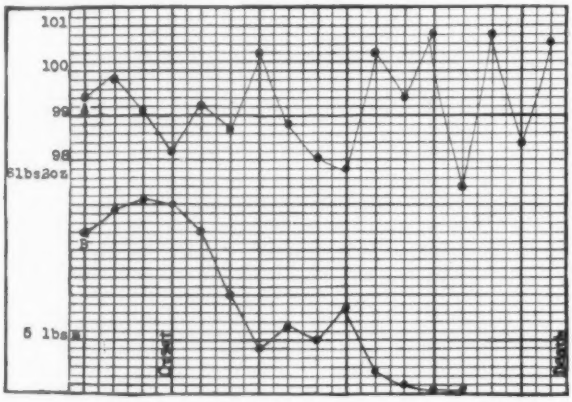
In the infants who died early in the course of the disease, the pathologic anatomic indications of enteritis were more marked and the mastoid lesions less advanced than in those who survived the first week or two of illness; later, the enteritis had usually subsided and the mastoid inflammation extended.

Routine bacteriologic examinations had been made at the autopsies on some of the first cases. These included cultures of the mastoid exudate on blood agar plates and of the heart's blood in broth. The cultures had shown the presence of colon bacilli, *Staphylococcus albus* and occasionally hemolytic streptococcus in the mastoid exudate. Colon bacilli had been isolated from the heart's blood in some cases.

After it became apparent that the deaths were due to an epidemic disease, a more complete bacteriologic investigation was undertaken.



A typical temperature and weight chart in a fatal case. *A* represents the temperature curve and *B*, the weight curve.

A study of the next cases that came to autopsy showed that there was a bacterial invasion of the blood stream by the same organisms found in the pus from the mastoid. These organisms were mostly colon bacilli and green-producing streptococci.

In order to determine whether the disease was primarily an infection of the upper respiratory tract causing otitis media and mastoditis or a primary infection of the alimentary tract resulting in a bacterial invasion of the blood stream and secondary mastoiditis, nose and throat cultures were made on blood agar plates from all sick and all well infants in the institution. The plates were incubated over night and examined the next day. A comparison of the bacteriology of the nose and throat in the sick and in the well infants did not show any difference in the two groups. Hemolytic streptococci were not present in the nose or throat cultures in either group. The throat cultures in all

showed green-producing streptococci predominating, with occasional staphylococci and pseudodiphtheria bacilli. The nose cultures showed chiefly staphylococci and pseudodiphtheria bacilli.

The green-producing streptococci found in cultures of the mastoid exudate in cases examined at post mortem were similar to those found in the throat cultures of both the sick and the well infants. Since both green-producing streptococci and the colon bacillus are normally present in infants, their presence in the exudates and blood stream at death did not furnish sufficient basis for the assumption that either was the primary cause of the epidemic. On the other hand, the fact that colon bacilli were found in cultures taken from the mastoid at operation in two cases, one four and the other six days before death, indicated that the invasion of the colon bacillus was not merely agonal. This suggested a possible intestinal origin of the infection. This view was supported by the clinical course of the disease which began with a characteristic diarrhea and by the pathologic observations which showed evidence of intestinal inflammation in twenty-five of the twenty-seven fatal cases. The fact that the earlier death occurred in the course of the disease the more marked were the indications of acute enteritis, such as hemorrhagic mucosa and swollen Peyer's patches, also pointed toward a primary intestinal infection.

At the next autopsies, additional cultures were made on lactose-litmus agar plates for the recognition of organisms of the typhoid, paratyphoid and dysentery groups. The Morgan dysentery bacillus was found in all of the five infants who died after this method of culture was adopted. In addition to the Morgan dysentery bacillus, *Proteus asiaticus* was isolated in case 23 and *Eberthella pyogenes* was found in cases 26 and 27. The last organism had been reported earlier in case 9. *Eberthella pyogenes* may have been present more frequently than it was recognized, because it produces acid in lactose and is not readily distinguished from the colon bacillus on a lactose medium.

The Morgan dysentery bacillus isolated was highly pathogenic for rabbits. From 1 to 3 cc. of a whole, forty-eight hour broth culture, injected intravenously, killed rabbits in from eight to forty-eight hours. From 3 to 5 cc. of sterile filtrate from forty-eight hour broth cultures, injected intravenously, killed rabbits within twenty-four hours. The rabbits which received the sterile filtrate and those that received the whole broth cultures showed hemorrhagic enteritis.

METHODS ADOPTED FOR THE CONTROL OF THE EPIDEMIC

As soon as it became apparent that we were not dealing with an air-borne disease but with an infection of intestinal origin, measures were adopted to safeguard the food of the infants, most of whom were at the time receiving various combinations of powdered milk prepara-

Summary of Fatal Cases

Case Number	Age at Onset of Disease	Clinical Symptoms	Operation	Day of Disease on Which Death Occurred	Anatomic Diagnosis	Bacteriology
1	21 days	Diarrhea; vomiting; no fever	None	2nd	Acute hemorrhagic enteritis; acute hyperplasia of mesenteric lymph glands; acute, fibrinopurulent peritonitis	No bacteriologic examination made
2	15 days	Diarrhea; loss of weight; irregular fever	Both ear drums punctured on 4th day and some pus obtained	15th	Bilateral, acute, suppurative otitis media and mastoiditis; acute, hemorrhagic enteritis and hyperplasia of the mesenteric lymph glands	No bacteriologic examination made
3	14 days	Diarrhea; progressive loss of weight; no fever till the 9th day	None	10th	Bilateral, acute, suppurative otitis media and mastoiditis; acute enterocolitis and hyperplasia of mesenteric lymph glands	No bacteriologic examination made
4	1 month	Diarrhea; loss of weight; irregular fever	Both ear drums incised on the 6th day without any drainage of pus	19th	Bilateral bronchopneumonia; bilateral, suppurative otitis media and mastoiditis; hyperplasia of mesenteric lymph glands	No bacteriologic examination made
5	15 days	Diarrhea; loss of weight; irregular fever	Both ear drums incised on the 3rd day without obtaining macroscopic pus	7th	Acute, hemorrhagic enteritis; hyperplasia of the mesenteric lymph glands; bilateral, acute catarrhal otitis media and mastoiditis	Cultures from both middle ears at operation showed Bacillus coli and Staphylococci
6	8 days	Diarrhea; progressive loss of weight; irregular fever; signs of pneumonia on 10th day; cough on 12th day	None	14th	Bilateral bronchopneumonia; bilateral, acute, suppurative otitis media and mastoiditis; acute enteritis and hyperplasia of mesenteric lymph glands	No bacteriologic examination made
7	17 days	Diarrhea; progressive loss of weight; no fever till 4th day	None	8th	Bilateral bronchopneumonia; acute enterocolitis; marked hyperplasia of mesenteric lymph glands	Cultures of blood from the heart showed Bacillus coli
8	16 days	Diarrhea; regurgitation; marked loss of weight; irregular fever	None	9th	Bilateral bronchopneumonia; bilateral, acute, suppurative otitis media and mastoiditis; acute, hemorrhagic enteritis with hyperplasia of Peyer's patches and mesenteric lymph glands	Cultures of heart's blood showed Bacillus coli; cultures of the mastoid exudate showed Bacillus coli, Staphylococcus albus and a few colonies of hemolytic streptococci

9	28 days	Diarrhea; vomiting; rapid loss of weight; no fever until the 5th day	None	10th	Bilateral, acute suppurative otitis media and mastoiditis; acute enteritis with hyperplasia of Peyer's patches, solitary lymph follicles and mesenteric lymph glands	Cultures of the heart's blood showed Eberthella pyogenes
10	14 days	Diarrhea; loss of weight; irregular fever beginning on the 4th day	None	7th	Left, acute, purulent otitis media and mastoiditis; acute enterocolitis; marked hyperplasia mesenteric lymph glands; uric acid infarcts in kidneys	Cultures of heart's blood showed Bacillus coli and Staphylococcus albus
11	20 days	Diarrhea; rapid loss of weight; low, irregular fever; dyspnea on 7th day	Both ear drums incised on 7th day without obtaining pus	8th	Bilateral bronchopneumonia; bilateral, acute, fibrinopurulent otitis media and mastoiditis	No bacteriologic examination made
12	5 weeks	Diarrhea; loss of weight, no fever, dyspnea on 11th day	None	12th	Bilateral bronchopneumonia; left suppurative otitis media and mastoiditis; hyperplasia of Peyer's patches and mesenteric lymph glands; ulceration of umbilical cord stump	Mastoid exudate contained Staphylococcus albus
13	12 days	Diarrhea and loss of weight without fever	Both ear drums incised and both mastoids drained on the 6th day; pus found in right mastoid; left mastoid appeared normal	7th	Bilateral bronchopneumonia; bilateral, acute, suppurative otitis media and mastoiditis; hyperplasia of mesenteric lymph glands	Bacillus coli was isolated from the heart's blood at autopsy and from the mastoid exudate before death
14	24 days	Diarrhea; loss of weight; no fever until the 8th day	Bilateral paracentesis on 7th day; double mastoidectomy on 11th day	17th	Bilateral, acute, suppurative otitis media and mastoiditis; acute enteritis and hyperplasia of mesenteric lymph glands	Cultures of mastoid exudate showed Bacillus coli, Staphylococcus albus and a few hemolytic streptococci
15	10 days	Diarrhea– progressive loss of weight; no fever till the 5th day	Double mastoidectomy on 3rd day and some gelatinous pus found on both sides	11th	Bilateral, acute, suppurative otitis media and mastoiditis	No bacteriologic examination made
16	18 days	Diarrhea; some loss of weight, sub normal temperature	None	6th	Bilateral, acute, suppurative otitis media and mastoiditis; acute enteritis and hyperplasia of mesenteric lymph glands	Bacillus coli isolated from heart's blood, and Bacillus coli and Staphylococcus albus found in mastoid exudate
17	15 days	Diarrhea; marked loss of weight; irregular fever	Both ear drums incised on 3rd day without obtaining pus	8th	Bilateral bronchopneumonia; hemorrhagic enteritis and hyperplasia of mesenteric lymph glands	No bacteriologic examination made
18	14 days	Diarrhea; marked loss of weight; irregular fever after 7th day; meningitis on 15th day	Both ear drums incised on 5th day	15th	Right acute mastoiditis and thrombosis of the right lateral sinus; acute hemorrhagic meningitis; acute enterocolitis and hyperplasia of mesenteric lymph glands	No bacteriologic examination made
19	7 weeks	Diarrhea; marked loss of weight; irregular fever	Both ear drums incised on 9th day without obtaining pus	10th	Bilateral bronchopneumonia; bilateral, acute, suppurative otitis media and mastoiditis with involvement of the petrous portion of the temporal bones; enteritis with some hemorrhagic areas in mucosa of ileum and hyperplasia of Peyer's patches	Mastoid exudate contained Bacillus coli, green-producing streptococcus, and an occasional Staphylococcus albus; cultures of heart's blood showed the same bacteriology

Summary of Fatal Cases—Continued

Case Number	Age at Onset of Disease	Clinical Symptoms	Operation	Day of Disease on Which Death Occurred	Anatomic Diagnosis	Bacteriology
20	2 weeks	Diarrhea; marked loss in weight; irregular fever after 9th day	Both ear drums incised on 5th day without obtaining pus	16th	Right, suppurative otitis media and mastoiditis, involving petrous portion of temporal bone; hemorrhagic enteritis with hyperplasia of Peyer's patches and mesenteric lymph glands	Cultures of heart's blood showed Bacillus coli, green-producing streptococcus, and the same organisms were found in the mastoid exudate
21	25 days	Diarrhea; rapid loss of weight; irregular fever after 5th day	None	8th	Bilateral, acute, suppurative otitis media and mastoiditis extending into petrous portion of temporal bones; acute enteritis with hyperplasia of Peyer's patches, solitary lymph follicles and mesenteric lymph glands	Cultures of the mastoid exudate and heart's blood showed Bacillus coli, green-producing streptococcus and Staphylococcus albus
22	3 months	Diarrhea; rapid loss of weight; irregular low fever	Double mastoidectomy on 10th day; no pus found	16th	Bilateral, catarrhal otitis media, hemorrhagic enteritis; enlargement of mesenteric lymph glands; acute pyelitis and cystitis; fibrous adhesions around the site of old operation for pyloric stenosis	Cultures of blood from mastoid bone at time of operation 6 days before death showed many colonies of Bacillus coli; no green-producing streptococci were found; cultures of heart's blood and middle ear cavities at autopsy showed Bacillus coli, green-producing streptococci and an occasional colony of staphylococcus
23	11 days	Diarrhea; loss of weight; irregular, low fever	Bilateral mastoidectomy done on 3rd day without finding any pus	7th	Bilateral bronchopneumonia; bilateral, slight otitis media; acute enteritis with hyperplasia of Peyer's patches and solitary lymph follicles; hyperplasia of mesenteric lymph glands	Cultures of blood obtained from mastoid bone at operation 4 days before death showed Bacillus coli; cultures from middle ear at autopsy showed Bacillus coli and green-producing streptococci; cultures of heart's blood made in broth and later plated out on lactose litmus agar showed Bacillus coli, Morgan dysentery bacillus, Proteus asiaticus, green-producing streptococci, and an occasional staphylococcus colony

24	18 days	Diarrhea; rapid loss of weight; irregular, low fever after 3rd day	None	7th	Right, acute, suppurative otitis media and mastoiditis; left, catarrhal otitis media; acute enteritis and hyperplasia of mesenteric lymph glands	Mastoid exudate contained Bacillus coli and green-producing streptococci Cultures of heart's blood plated out on lactose litmus agar and on blood agar showed Bacillus coli, Morgan dysentery bacillus, and green-producing streptococci
25	5 months	Diarrhea; loss of weight; irregular fever after 3rd day	None	8th	Bilateral, acute, catarrhal otitis media and mastoiditis; acute enteritis with hyperplasia of Peyer's patches, solitary lymph follicles and mesenteric lymph glands	Cultures of mastoid exudate at autopsy showed Bacillus coli and green-producing streptococci; cultures of heart's blood plated out on lactose litmus agar and blood agar showed Bacillus coli, green-producing streptococci and Morgan dysentery bacillus; cultures of the mucosa of the ileum on lactose-fuchsin agar showed Bacillus coli and Morgan dysentery bacillus
26	13 days	Diarrhea; loss of weight; irregular, low fever	None	7th	Bilateral bronchopneumonia; bilateral, acute, suppurative otitis media with mastoiditis involving the petrous portion of the temporal bones; marked, acute hemorrhagic enterocolitis with hyperplasia of Peyer's patches and solitary lymph follicles; hyperplasia of mesenteric lymph glands	Cultures of intestinal mucosa and mesenteric lymph glands showed Bacillus coli, Eberthella pyogenes and some Morgan dysentery bacilli; cultures of mastoid exudate, heart's blood and lung showed Bacillus coli, Eberthella pyogenes and green-producing streptococci
27	9 days	Diarrhea; continued loss of weight; irregular, low fever; dyspnea on 11th day	None	12th	Bilateral bronchopneumonia; bilateral, acute, suppurative otitis media and mastoiditis; marked acute, hemorrhagic enterocolitis with enlargement of Peyer's patches and hyperplasia of the mesenteric lymph glands	Cultures of the intestinal mucosa and mesenteric lymph glands on lactose, litmus and blood agar showed Bacillus coli, Eberthella pyogenes, and Morgan dysentery bacilli; cultures of the mastoid exudate and heart's blood showed Bacillus coli, Eberthella pyogenes and green-producing streptococci

tions. These powdered milk preparations were cultured and found to contain a variety of living bacteria.[1] It was undesirable to boil the powdered milk feedings, on account of their tendency to curdle. But, since the infection was introduced through the mouth, it was important to give only food that could be sterilized by boiling and to give the food as fresh as possible, so that there would be little chance for the production of toxic substances that might not be destroyed by heat. To accomplish this, the powdered milk preparations were discarded, and only dilutions of fresh breast milk, fresh cow's milk or protein milk freshly prepared in the diet kitchen were given. All feedings and all water were boiled ten minutes. The bottles and nipples were autoclaved.

In the care of infants, conditions are particularly favorable for transmission of intestinal infections by the fingers, because the same nurses who bathe the babies and change the diapers usually give the feedings. It is conceivable that the hands, which may become thoroughly contaminated in changing the diapers of an infected baby, might, in spite of washing, contaminate the feeding of another baby in various ways, as by handling the nipple while placing it on the bottle. To avoid transmission of the infection by the fingers, nurses were required to put on a fresh pair of sterile rubber gloves for each feeding of each baby and for giving water. Precautions were also taken in the diet kitchen and throughout the institution so that no finger was allowed to come in contact with anything that went into a baby's mouth. Diapers of all infants were put in lysol solution immediately on removal.

The results of changing the feeding to boiled fresh milk preparations and the adoption of precautions to eliminate the possibility of contamination by the fingers of the nurses were apparent at once in a decrease in sickness among the infants. The epidemic came to an abrupt end.

COMMENT

The epidemic disease reported is interesting because it was due to a primary intestinal infection with the Morgan dysentery bacillus and was regularly complicated by otitis media and mastoiditis. Whether *Eberthella pyogenes* and *Proteus asiaticus*, isolated in some of the cases, took part in the primary infection is not determined. In the more chronic cases, the subsidence of the acute intestinal inflammation and the progression of the mastoid lesions which resulted in necrosis of the petrous portion of the temporal bone or sinus thrombosis and meningitis obscured the intestinal origin of the disease.

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Epidemics of diarrhea among infants similar to the one reported in their clinical manifestations and characterized by the development of mastoiditis have been reported recently by Marriott,[2] Alden,[3] Lyman,[4] and Sidbury,[5] who refer to the earlier reports by Hartmann,[6] Preysing[7] and Renaud.[8] The bacteriologic examinations in the cases previously described have been largely confined to cultures of the middle ear and mastoid pus without special reference to the intestinal bacteriology or organisms of the *Eberthella* and *Salmonella* groups and without the use of cultural methods adequate for the detection of these organisms.

CONCLUSION

That this epidemic of enteritis associated with mastoiditis in infants was primarily an intestinal infection is shown by the results of bacteriologic examinations in which the Morgan dysentery bacillus was found and by the fact that the epidemic was controlled through discarding all foods which were not fresh and which could not be boiled and the adoption of measures to prevent the transfer of intestinal bacteria by the fingers of nurses.

It has been demonstrated that the occurrence of mastoiditis as well as the enteritis to which it was secondary is prevented by the prophylactic measures adopted; that the morbidity from all causes has been reduced to a minimum is shown by the fact that no instance of sickness of any kind occurred in the institution in the two and a half months following the adoption of the technic described.

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3. Alden, A. M.: Gastro-Intestinal Disturbances in Infants as a Result of Streptococcus Infection in Ears, South. M. J. **19**:360 (May) 1926.

4. Lyman, H. W.: Mastoiditis as the Cause of Gastro-Intestinal Disturbances in Infants, J. Missouri M. A. **22**:293 (Aug.) 1925.

5. Sidbury, J. B.: Mastoiditis in Infants: Report of Forty Operated Cases, South. M. J. **20**:713 (Sept.) 1927.

6. Hartmann: Bie Einwirkung der Otitis Media de Säuglinge auf den Verdauungs apparat, Ztschr. f. Ohrenh. **34**:1, 1898.

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THE SEX FACTOR IN INFANTILE TETANY*

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It is well known that a great many diseases show a marked sex predisposition. This is true not only of the ailments of adults but of those of children. Thus, pyloric stenosis is from five to six times as common in boys as in girls. Chorea is more than twice as frequent in girls as in boys. Congenital laryngeal stridor is said to be more common in males.[1] Such knowledge is often of diagnostic and prognostic value. It has, however, another significance: when environmental conditions for the two sexes are uniform, it indicates, in infants at any rate, a constitutional predisposition for the particular disease, which may be an aid in evaluating the factors that determine its occurrence.

The relationship of sex to infantile tetany has received little attention in the literature. Most textbooks and monographs do not mention the subject. Griffith [2] states that the condition is more common in boys than in girls. Howard,[3] in a series of forty-seven cases, found males more susceptible (thirty-three boys to fourteen girls). Laryngologists have long recognized the predominance of males among infants with laryngismus stridulus. In 1880, Morell Mackenzie [4] found this disease more than twice as often in boys as in girls. In mortality statistics of the United States for the past ten years, the ratio of deaths from infantile convulsions in male infants to those in female infants, all under 1 year of age, is consistently higher than the ratio of deaths in males to those in females of the same age from all causes.

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* Received for publication, Dec. 19, 1927.

* From the Department of Diseases of Children, College of Physicians and Surgeons, Columbia University; the Children's Medical Division and the Department of Pathology, Bellevue Hospital.

1. Thomson, St. Clair: Diseases of Nose and Throat, ed. 3, New York, 1926, p. 503.

2. Griffith, J. P. C.: Diseases of Infants and Children, Philadelphia, W. B. Saunders Company, 1921, vol. 2, p. 251.

3. Howard, C. P.: Tetany: A Report of Nine Cases, Am. J. M. Sc. **131**:301, 1906.

4. Mackenzie, M.: Diseases of the Throat and Nose, 1880, vol. 1, p. 463.

During the past two years, studies have been made of the factors which lead to tetanic hypocalcemia in infants. In this report, discussion will be limited to sex as a predisposing factor.

The material consists of seventy cases of infantile tetany from the Children's Medical Division of Bellevue Hospital. The diagnosis was made from the clinical manifestations, determinations of serum calcium, and, in many instances, the electrical reactions. Through the kindness of Dr. Herbert B. Wilcox, the records of sixty-six patients from the wards of the Babies' Hospital, New York, have been examined and added to our own, making a total of 136. In the latter cases, the clinical diagnosis was verified, except in a few instances, by estimations of serum calcium. The patients ranged in age from 5 weeks to 26 months.

The sex distribution for tetany in infants is shown in table 1. Of 136 patients, ninety-three, or 68 per cent, were boys and forty-three, or 32 per cent, girls. A similar proportion of boys to girls (eighteen to

TABLE 1.—*The Sex Distribution for Tetany in Infants*

Source (Hospital)	Total Number of Cases	Boys	Girls
Bellevue	70	47	23
Babies'	66	46	20
Total	136	93	43
Per cent	100	68	32

eight) was found in cases of tetany from the outpatient department of the Babies' Hospital. In this series, therefore, infantile tetany was more than twice as common in boys as in girls. This indicates a predisposition on the part of males for tetany comparable to that of females for chorea (71 per cent girls, according to Osler).

When the cases of tetany are divided into two groups, one group including babies 8 months of age and below, and the other including babies above 8 months of age, it is found that the predominance of boys is more marked during the first eight months of life. Thus, of the 136 children in this series, the proportion of boys to girls in the younger group was sixty-nine to twenty-three, while in the group above 8 months the proportion was only twenty-three to nineteen. Sex is, therefore, of importance, not only as a factor determining which infants will get tetany, but as a factor determining at what age the child will develop it.

Of the seventy patients studied at Bellevue Hospital, tetany was in large part responsible for the deaths of nine. All were boys. A résumé of the cases is given in table 2. With one exception, death occurred within forty-eight hours after admission to the hospital. Eight of the

nine patients had severe convulsions, which were associated with pneumonia in six instances. The patient who did not have convulsions—an infant with pneumonia and congenital syphilis—was found dead in his crib. The increased severity of tetany in male infants is striking.

The average serum calcium concentration for both sexes was the same (6.55 mg. per hundred cubic centimeters of serum for boys; 6.59 mg. for girls). The greater severity of the disease in boys is, therefore, not dependent on lower calcium values in the serum.

TABLE 2.—*Deaths of Infants with Tetany*

Patient	Age, Months	Date of Admission	Length of Stay in Hospital	Mg. of Serum Calcium	Mg. of Inorganic Phosphorus	Autopsy Performed	Comment
J. E.....	5/4	6/ 1/26	15 hr.	7.2	6.2	No	Died in convulsion; marked laryngospasm
S. P.....	2	1/10/27	48 hr.	6.2	9.0	Yes	Pneumonia; severe convulsions lasting several hours
H. H.....	2	1/27/27	12 hr.	5.9	3.3	Yes	Died suddenly; pneumonia and congenital syphilis; thymus and lymphatic apparatus hypertrophied
S. S.....	3	12/29/26	15 hr.	6.7	5.6	No	Marked laryngospasm; convulsions; died in cyanotic attack
F. P.....	5	1/24/27	20 hr.	6.1	6.7	Yes	Severe convulsions lasting several hours; temperature before death, 109.4 F.; pneumonia; status lymphaticus
J. M.....	7	4/ 1/27	7 days	6.3	3.5	Yes	Severe convulsions on admission, lasting several hours; pneumonia
F. C.....	7	4/12/27	36 hr.	7.2	6.2	No	Admitted to hospital with convulsions; desperately ill; pneumonia; temperature, 108.2 F.
W. B.....	11	5/ 5/27	36 hr.	5.6	3.1	Yes	Admitted with convulsions; desperately ill; pneumonia
T. C.....	14	11/21/26	24 hr.	5.6	...	Yes	Died in convulsions, autopsy negative

The greater tendency on the part of boys to develop tetanic hypocalcemia represents a physiologic difference in the calcium regulating mechanism of the two sexes during infancy. This may account, in part at least, for the higher ratio of deaths in male infants to deaths in female infants during the winter and early spring months. For example, in the month of April, 1922, there were 140 deaths in male infants under 1 year as compared to 100 deaths in females, while in the summer of the same year this ratio dropped to 128: 100. This will be discussed more fully in a later publication.[5]

5. Bakwin, H.: The Sex Factor in Infant Mortality, read before the Pediatric Section, New York Academy of Medicine, March, 1928.

SUMMARY

1. In a series of 136 patients with infantile tetany, boys were affected more than twice as often as girls (93:43).

2. The preponderance of males susceptible to this disease is particularly striking during the first eight months of life.

3. The disease is apt to be more severe in boys than in girls. The nine patients whose death was in large part due to tetany were boys

4. The more frequent occurrence of tetany in boys indicates a difference in the calcium regulating mechanism of the two sexes.

METABOLISM OF UNDERNOURISHED CHILDREN

V. PROTEIN METABOLISM *

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In earlier papers in this series, we have reported investigations of various phases of the metabolism of undernourished children.[1] In this paper, we give the results of fifty-nine sets of experiments on the nitrogen metabolism of ten normal and thirty-nine underweight children, most of whom have served as subjects in our previous studies. Little literature is available on this subject. The only article which gives data having a direct bearing on our problem is that of Fleming and Hutchison,[2] who, in a study of the metabolism of undernourished infants, reported that the normal absorption of nitrogen is from 80 to 95 per cent of the intake and that a difference was not found in the percentage of nitrogen absorbed in undernourished infants. These investigators also found that with improvement in the nutritional state, an increase in the output of urinary nitrogen occurred. In other words, as the infant gained in weight the percentage of nitrogen retained in the body decreased.

EXPERIMENTAL

The same technic of feeding and of collection and analysis of food and excreta was used in this series of experiments as has already been reported in previous papers.[3] As in our previous work, the children have been divided into five groups, as follows: group 1, vigorous nor-

* Received for publication, Jan. 17, 1928.

* From the Nelson Morris Institute for Medical Research and the Sarah Morris Hospital for Children of the Michael Reese Hospital. Aided by a fund contributed jointly by the Elizabeth McCormick Fund, Mrs. Francis Neilson, Mrs. Gusta Morris Rothschild and Mr. Melville M. Rothschild.

1. Wang, C. C.; Frank, M.; Hays, B.B., and Kern, R.: Metabolism of Undernourished Children: I. Gain in Weight, Am. J. Dis. Child. **32**:63 (July) 1926; Metabolism of Undernourished Children: II. Basal Metabolism, ibid. **32**: 350 (Sept.) 1926; Metabolism of Undernourished Children: III. Urinary Nitrogen with Special Reference to Creatinine, ibid. **32**:360 (Sept.) 1926. Wang, C. C.; Kaucher, M., and Frank, M.: Metabolism of Undernourished Children: IV. Calcium Metabolism, ibid. **35**:856 (May) 1928.

2. Fleming, G. B., and Hutchison, H. S.: A Study of the Metabolism in the Undernourished Infant, Quart. J. Med. **68**:339 (July) 1924.

3. Wang, Frank, Kern and Hays (footnote 1, third reference). Footnote 2

TABLE 1.—*Nitrogen Metabolism of Vigorous Normal Children (Group 1)*

Subject	Age	Sex*	Weight		Nitrogen Intake		Nitrogen Output Per 24 Hours				Absorption			Retention		
			Kilograms	Per Cent Variation from Normal	Per 24 Hours (Gm.)	Per Kilogram Per 24 Hours (Gm.)	Urine (Gm.)	Feces (Gm.)	Total (Gm.)	Total Per Kilogram (Gm.)	Total (Gm.)	Per Kilogram (Gm.)	Per Cent of Intake	Total (Gm.)	Per Kilogram (Gm.)	Per Cent
M. S.	9	♂	33.5	+17.1	13.3	0.396	10.1	1.13	11.2	0.334	12.2	0.363	91.5	1.99	0.059	15.0
M. F.	8	♀	27.7	+ 8.6	11.3	0.409	9.4	1.07	10.5	0.378	10.3	0.371	90.6	0.86	0.031	7.6
L. F.	10	♀	26.9	+ 3.9	12.1	0.450	10.5	0.81	11.3	0.421	11.3	0.420	93.3	0.77	0.029	6.4
M. H.	9	♀	23.9	+ 3.2	12.8	0.536	10.9	1.10	12.0	0.502	11.7	0.470	91.4	0.80	0.034	6.3
S. A.	10	♀	29.2	+ 1.4	13.1	0.448	11.3	1.01	12.3	0.419	12.1	0.413	92.3	0.85	0.029	6.5
L. L.	10	♂	34.0	+ 1.2	13.4	0.396	10.7	0.92	11.6	0.343	12.5	0.368	93.1	1.78	0.053	13.3
B. L.	12	♀	33.0	+ 1.2	15.2	0.462	12.4	1.12	13.6	0.411	14.1	0.428	92.6	1.68	0.051	11.0
F. J.	11	♀	28.2	+ 0.6	12.9	0.457	10.6	1.04	11.6	0.411	11.8	0.420	92.2	1.28	0.045	9.9
R. G.	11	♀	28.6	— 3.4	12.9	0.450	10.5	1.15	11.7	0.409	11.7	0.410	90.8	1.19	0.042	9.1
H. S.	9	♀	27.2	— 3.5	12.8	0.471	10.7	1.12	11.8	0.434	11.7	0.430	91.4	1.00	0.037	7.8
Average	9.9	..	....	+ 3.0	....	0.448	....	....	....	0.406	....	0.409	91.9	....	0.041	9.3

* In this table and in tables 2, 3, 4 and 5, ♂ indicates male; ♀, female.

TABLE 2.—*Nitrogen Metabolism of Children Normal in Weight Only (Group 2)*

Subject	Age	Sex	Weight		Nitrogen Intake		Nitrogen Output Per 24 Hours				Absorption			Retention		
			Kilograms	Per Cent Variation from Normal	Per 24 Hours (Gm.)	Per Kilogram Per 24 Hours (Gm.)	Urine (Gm.)	Feces (Gm.)	Total (Gm.)	Total Per Kilogram (Gm.)	Total (Gm.)	Per Kilogram (Gm.)	Per Cent of Intake	Total (Gm.)	Per Kilogram (Gm.)	Per Cent
E. F.	6	♀	19.3	+ 4.9	10.0	0.518	8.0	0.89	8.9	0.461	9.1	0.472	91.0	1.10	0.057	11.0
R. M.	5	♂	14.4	± 0.0	9.1	0.632	6.3	1.02	7.3	0.507	8.1	0.563	89.0	1.80	0.125	19.8
H. D.	5	♂	13.8	± 0.0	7.9	0.573	5.9	0.80	6.7	0.486	7.1	0.515	89.9	1.20	0.087	15.2
J. G.	8	♂	23.8	± 0.0	12.4	0.521	9.7	0.98	10.7	0.450	11.4	0.479	91.9	1.70	0.071	13.7
E. G.	6	♀	18.1	— 1.1	8.9	0.492	6.8	0.81	7.6	0.420	8.1	0.448	91.0	1.30	0.072	14.6
N. G.	7	♂	16.9	— 1.2	9.8	0.580	7.5	0.90	8.4	0.497	8.9	0.527	90.8	1.40	0.083	14.3
E. G.	6	♀	17.5	— 1.7	8.8	0.503	6.3	0.87	7.2	0.411	7.9	0.451	89.8	1.60	0.091	18.2
G. L.	13	♀	34.7	— 3.3	9.8	0.282	7.1	1.00	8.1	0.233	8.8	0.254	89.8	1.70	0.049	17.3
N. G.	7	♂	16.5	— 3.5	9.7	0.588	6.5	0.86	7.4	0.449	8.8	0.533	90.7	2.30	0.139	23.7
K. B.	8	♀	20.7	— 3.7	12.1	0.585	9.3	0.87	10.2	0.580	11.2	0.541	92.6	1.90	0.092	15.7
E. G.	6	♀	17.5	— 1.7	8.7	0.497	5.9	0.79	6.7	0.383	7.9	0.451	90.8	2.00	0.114	23.0
I. Ba.	10	♂	30.5	— 4.1	11.3	0.366	7.3	1.14	8.4	0.275	10.5	0.340	92.9	2.89	0.094	25.6
E. F.	6	♀	17.3	— 4.9	9.9	0.572	7.7	1.06	8.8	0.509	8.8	0.509	88.9	1.10	0.064	11.1
Average	7.2	..	....	— 1.6	....	0.516	....	....	....	0.431	....	0.468	90.1	....	0.088	17.2

mal children; group 2, those normal in weight only; group 3, those from 5.1 to 10.0 per cent underweight; group 4, those from 10.1 to 15.0 per cent underweight; group 5, those more than 15 per cent underweight.

COMMENT

Although the effort was made to put all the children on the same intake of nitrogen, computed on the basis of body weight, this was found to be impossible, owing to the fact that some of the children refused to eat the quantity of food offered, while others displayed a ravenous appetite. Except in three instances, the intake varied from 0.396 to 0.687 Gm. of nitrogen per kilogram of body weight, corresponding to a protein intake of from 2.48 to 4.29 Gm. per kilogram. The lowest intake of 0.282 Gm. per kilogram occurred in a girl, aged 13 years, who was only 3.3 per cent underweight. Another instance of low intake also occurred in a child who was normal in weight but not in appearance. The average intake of each group increased with the degree of undernutrition. This is probably due to the fact that the nitrogen content of the diet was calculated according to what the protein intake should be if the child were of normal weight for its height and age.

In agreement with Fleming and Hutchison, we found that the percentage of nitrogen absorbed was practically the same in all five groups, regardless of the degree of undernutrition. Average absorption for the five groups varied from 90.1 to 91.9 per cent of the intake.

A decided increase in nitrogen retention was observed among the undernourished children, both in percentage of retention and in retention per kilogram of body weight. In the normal group the percentage of retention ranged between 6.3 and 15.0, with an average of 9.3, whereas the minimum retention of the second group was 11.0 per cent and the maximum 25.6 per cent, with an average of 17.2 per cent. The increase in percentage of retention in the next three groups, though striking when compared with the vigorous normal group, is not so marked in comparison with the group normal in weight only, the average percentage of retention being 20.3, 22.1 and 24.3 for groups 3, 4 and 5, respectively.

The marked difference in retention between the vigorous normal children and those normal in weight only may be due to several possible factors. In the first place, the average age in group 2 (normal in weight only) is only 7.2 years, while the average age for group 1 (vigorous normals) is 9.9 years. It has been generally known that the younger the child the greater the retention. In the second place, a number of the subjects in the second group had been in the hospital for a comparatively long time and had been going through a period of rapid gain in weight. Also, there is a possibility that the children in group 2 were to some extent underheight for their age, although not underweight for

TABLE 3.—*Nitrogen Metabolism of Children from 5.1 to 10.0 Per Cent Underweight (Group 3)*

Subject	Age	Sex	Weight		Nitrogen Intake		Nitrogen Output Per 24 Hours				Absorption			Retention		
			Kilograms	Per Cent Variation from Normal	Per 24 Hours (Gm.)	Per Kilogram Per 24 Hours (Gm.)	Urine (Gm.)	Feces (Gm.)	Total (Gm.)	Total Per Kilogram (Gm.)	Total (Gm.)	Per Kilogram (Gm.)	Per Cent of Intake	Total (Gm.)	Per Kilogram (Gm.)	Per Cent
T. F.	10	♂	24.0	— 5.3	11.7	0.458	8.6	0.96	9.6	0.400	10.7	0.446	91.5	2.10	0.088	17.9
E. B.	5	♀	15.8	— 5.4	8.7	0.551	5.4	0.89	6.2	0.392	7.9	0.500	90.8	2.50	0.158	28.7
J. G.	8	♂	22.4	— 5.9	10.5	0.469	7.4	0.89	8.3	0.371	9.6	0.429	91.4	2.20	0.098	21.0
A. Go	10	♀	27.6	— 6.1	11.0	0.399	8.7	0.93	9.6	0.348	10.1	0.363	91.8	1.40	0.051	12.7
R. G.	6	♂	16.3	— 7.9	9.3	0.571	6.3	0.69	7.0	0.429	8.6	0.528	92.5	2.30	0.141	24.7
L. C.	6	♀	18.7	— 7.9	9.3	0.514	6.4	0.72	7.1	0.392	8.6	0.476	92.5	2.20	0.122	23.7
E. D.	10	♂	25.0	— 8.1	11.5	0.460	8.9	1.04	9.9	0.396	10.5	0.429	91.3	1.60	0.064	13.9
J. U.	9	♀	24.9	— 8.2	10.7	0.415	6.2	0.81	7.0	0.270	9.9	0.334	92.5	3.74	0.144	34.8
E. G.	6	♀	16.3	— 8.4	9.5	0.583	6.4	1.00	7.4	0.454	8.5	0.521	89.5	2.10	0.129	22.1
B. G.	9	♀	20.5	— 8.5	10.5	0.512	7.7	0.70	8.4	0.410	9.8	0.478	93.3	2.10	0.102	20.0
H. K.	10	♂	21.7	— 8.8	13.7	0.631	10.7	1.02	11.7	0.539	12.7	0.585	92.7	2.00	0.092	14.6
H. D.	5	♂	13.2	— 9.4	7.0	0.530	5.1	0.60	5.7	0.432	6.4	0.485	91.4	1.30	0.098	18.6
L. C.	6	♀	18.4	— 9.4	9.1	0.495	6.7	0.68	7.4	0.402	8.4	0.457	92.3	1.70	0.092	18.7
C. S.	10	♂	27.0	— 9.4	11.1	0.415	6.5	1.03	7.5	0.277	10.1	0.376	90.7	3.59	0.133	32.3
S. K.	10	♂	27.5	— 9.8	14.5	0.527	11.4	1.07	12.5	0.455	13.4	0.487	92.4	2.00	0.073	13.8
T. F.	10	♂	23.4	— 9.8	12.8	0.547	9.5	0.98	10.5	0.449	11.8	0.504	92.2	2.30	0.098	18.0
Average	8.1	..	....	— 8.0	....	0.505	....	....	....	0.401	....	0.462	91.8	....	0.105	20.3

TABLE 4.—*Nitrogen Metabolism of Children from 10.1 to 15.0 Per Cent Underweight (Group 4)*

Subject	Age	Sex	Weight		Nitrogen Intake		Nitrogen Output Per 24 Hours				Absorption			Retention		
			Kilograms	Per Cent Variation from Normal	Per 24 Hours (Gm.)	Per Kilogram Per 24 Hours (Gm.)	Urine (Gm.)	Feces (Gm.)	Total (Gm.)	Total Per Kilogram (Gm.)	Total (Gm.)	Per Kilogram (Gm.)	Per Cent of Intake	Total (Gm.)	Per Kilogram (Gm.)	Per Cent
M. S.	9	♀	21.6	—10.4	10.8	0.500	7.6	0.89	8.5	0.394	9.9	0.428	91.7	2.30	0.106	21.3
H. L.	8	♀	19.3	—10.6	9.7	0.503	6.8	1.11	7.9	0.409	8.6	0.446	88.7	1.80	0.093	18.6
H. G.	11	♂	27.5	—10.7	11.7	0.425	8.3	1.08	9.4	0.342	10.6	0.386	90.6	2.30	0.084	19.7
I. Bo	9	♀	20.7	—10.7	9.8	0.471	5.8	1.05	6.9	0.333	8.7	0.420	89.2	2.91	0.141	29.8
L. C.	6	♀	18.0	—11.3	9.4	0.522	6.6	0.69	7.3	0.406	8.7	0.483	92.6	2.10	0.117	22.3
M. M.	7	♂	17.9	—11.8	9.3	0.520	6.1	0.92	7.0	0.391	8.4	0.469	90.3	2.30	0.128	24.7
B. B.	5	♀	12.0	—11.8	8.2	0.683	5.8	0.77	6.6	0.550	7.4	0.617	90.2	1.60	0.133	19.5
S. K.	10	♂	26.8	—12.1	15.9	0.593	12.3	1.10	13.4	0.500	14.8	0.552	93.1	2.50	0.093	15.7
J. M.	13	♂	27.5	—12.1	12.5	0.455	9.4	0.94	10.3	0.375	11.6	0.422	92.8	2.20	0.080	17.6
R. G.	10	♀	19.1	—12.3	9.2	0.482	6.0	0.86	6.9	0.361	8.3	0.437	90.7	2.31	0.121	25.1
N. G.	7	♂	15.0	—12.3	10.3	0.687	7.0	0.96	8.0	0.533	9.3	0.620	90.3	2.30	0.153	22.3
F. B.	10	♀	17.8	—13.2	11.4	0.640	9.0	0.80	9.8	0.551	10.6	0.596	93.0	1.60	0.090	23.3
E. F.	6	♀	15.4	—13.5	9.2	0.597	6.6	0.70	7.3	0.474	8.5	0.552	92.4	1.90	0.123	20.7
B. G.	8	♀	19.1	—13.6	10.0	0.523	5.8	1.05	6.8	0.356	8.9	0.468	89.5	3.16	0.166	31.6
R. B.	6	♂	16.4	—14.1	9.1	0.555	6.5	0.91	7.4	0.451	8.2	0.500	90.1	1.70	0.104	18.7
Average	8.3	..	....	—12.0	....	0.544	....	....	....	0.428	....	0.495	91.0	....	0.115	22.1

TABLE 5.—*Nitrogen Metabolism of Children More than 15 Per Cent Underweight (Group 5)*

Subject	Age	Sex	Weight		Nitrogen Intake		Nitrogen Output Per 24 Hours				Absorption			Retention		
			Kilograms	Per Cent Variation from Normal	Per 24 Hours (Gm.)	Per Kilogram Per 24 Hours (Gm.)	Urine (Gm.)	Feces (Gm.)	Total (Gm.)	Total Per Kilogram (Gm.)	Total (Gm.)	Per Kilogram (Gm.)	Per Cent of Intake	Total (Gm.)	Per Kilogram (Gm.)	Per Cent
L. C.	6	♀	17.1	—15.8	8.4	0.491	5.0	0.65	5.7	0.333	7.7	0.450	91.6	2.70	0.158	32.1
D. Y.	7	♀	17.3	—16.0	10.2	0.590	7.0	0.96	8.0	0.462	9.2	0.532	90.2	2.20	0.127	21.6
E. F.	6	♀	15.2	—16.5	9.2	0.605	6.1	0.89	7.0	0.461	8.3	0.546	90.2	2.20	0.145	23.9
S. K.	10	♂	25.4	—16.7	15.3	0.602	11.9	1.01	12.9	0.508	14.3	0.563	93.5	2.40	0.094	15.7
A. S.	10	♀	17.8	—16.8	11.9	0.669	8.2	1.00	9.2	0.517	10.9	0.612	91.6	2.70	0.152	22.7
L. B.	11	♀	25.8	—17.8	13.4	0.519	9.6	1.20	10.8	0.419	12.2	0.473	91.0	2.60	0.101	19.4
I. Be.	11	♀	26.0	—17.2	9.7	0.373	5.8	1.03	6.8	0.262	8.7	0.335	89.7	2.90	0.116	29.9
A. B.	6	♀	16.9	—18.0	9.6	0.568	6.8	0.79	7.6	0.450	8.8	0.521	91.7	2.00	0.118	20.8
A. S.	10	♀	17.4	—18.7	10.0	0.575	7.0	0.80	7.8	0.448	9.2	0.529	92.0	2.20	0.126	22.0
A. S.	10	♀	16.9	—19.2	10.4	0.615	7.3	0.81	8.1	0.479	9.6	0.568	92.3	2.30	0.136	22.1
B. B.	5	♀	11.2	—19.3	7.4	0.661	4.8	0.64	5.4	0.482	6.8	0.607	91.9	2.00	0.179	27.0
L. P.	8	♀	22.6	—19.8	10.1	0.447	6.6	1.00	7.6	0.336	9.1	0.403	90.1	2.5	0.111	24.7
A. G.	10	♀	20.6	—23.4	13.7	0.665	8.4	1.10	9.5	0.461	12.6	0.612	91.8	4.2	0.204	30.7
R. N.	11	♀	25.2	—25.0	10.8	0.429	7.4	0.79	8.2	0.325	10.0	0.397	92.6	2.6	0.103	24.1
A. G.	8	♀	17.9	—25.7	10.6	0.592	6.6	1.00	7.6	0.425	9.6	0.536	90.6	3.0	0.168	28.3
Average	8.6	..	....	—19.1	....	0.560	....	....	....	0.425	....	0.512	91.4	....	0.134	24.3

their height, and therefore were still growing more rapidly than the normal children.

The average retention per kilogram of body weight increased from 0.041 Gm. of nitrogen for the vigorous normal children to 0.134 for the group of children more than 15 per cent underweight, corresponding to from 0.26 to 0.84 Gm. of protein per kilogram in twenty-four hours.

The results of this investigation would seen to show that the ability of the undernourished child to absorb and store nitrogen is fully equal to, or greater than, that of the normal child. The fact that the nitrogen retention increases with the degree of underweight indicates that as the child gains in weight he is building muscular tissue as well as storing fat. Our failure to observe a similar increase in calcium retention among the underweight children may be explained either on the ground that the underweight children may not have been growing any more rapidly than were the normal children, or by the facts that bone growth goes on much more slowly than does the growth of muscular tissue, and that our experiments could not be continued over a long enough period to observe any marked calcium retention.

The question may arise as to why an increase in basal metabolism was not noted when the children were storing nitrogen at such a rapid rate. If one assumes that 15 per cent of the body weight is protein and uses the maximum value, 0.84 Gm. of protein retained by one of the undernourished children, a child weighing 20 Kg. would be gaining at a rate of about 112 Gm. in twenty-four hours. Since the average energy production of a child of 8 years is about 38 calories per kilogram per day, it will be seen that the increase in energy production due to the storage of protein would be only about 4 calories a day, which is negligible.

Because of the storage of nitrogen which we observed in our underweight children, it is evident that the protein requirement of these children is greater than that of normal children. Therefore, in computing diets for underweight children, the protein as well as the caloric intake should be computed according to the standard, rather than to the actual, weight of the child in question.

SUMMARY

In a study of the nitrogen metabolism of ten normal and thirty-nine underweight children, it was found that while the percentage of absorption was practically the same for both the normal and the undernourished children, the percentage of nitrogen retained in the body as well as the nitrogen retention per kilogram of body weight increased with the degree of undernutrition.

It is suggested that the protein requirement of underweight children should be computed according to the standard, rather than to the actual, weight of the child.

SCARLET FEVER

VI. THE RELATION BETWEEN THE LOSS OF SKIN SENSITIVITY TO TOXIN AND THE DEVELOPMENT OF ANTITOXIC IMMUNITY DURING ARTIFICIAL IMMUNIZATION *

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The active immunization against scarlet fever by repeated injections at weekly intervals of scarlatinal streptococcus filtrate toxin as described by Dick and Dick [1] has been reported by a number of observers. It is said that following such injections an immunity usually develops which lasts from several months to a year and a half or possibly longer. The assumption of immunity is based not only on a decreased incidence of clinical scarlet fever in such injected persons, but chiefly on the fact that the reaction to the Dick test, or skin sensitivity to a small amount of toxin, is rendered negative by the injections of toxin. This loss of sensitivity of the skin has been thought to depend on the production of a specific scarlatinal antitoxin, such as occurs in patients convalescing from cases of clinical scarlet fever.

In a previous paper,[2] it was suggested that the skin reaction to injected filtrate toxin is an evidence of specific anaphylactic hypersusceptibility to a streptococcus protein. In support of this view, instances were reported [3] in which skin sensitivity was apparently induced passively in certain nonsensitive infants by the injection of serum from a sensitive person. The possibility that evidence of desensitization might appear as a result of injections of toxin into sensitive persons suggested a more intensive study of this phenomenon. In the present paper are reported the changes in skin sensitivity which take place after the injection of toxin into very sensitive persons, and their relation to the development of antitoxin in the blood.

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* Received for publication, Jan. 9, 1928.

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1. Dick, G. F., and Dick, G. H.: Scarlet Fever Toxin in Preventive Immunization, J. A. M. A. **82**:544 (Feb. 16) 1924.

2. Cooke, J. V.: Scarlet Fever: II. The Development of Toxin Sensitivity of the Skin in Infants and Its Relation to the Presence of Antitoxin in the Blood, Am. J. Dis. Child. **35**:762 (May) 1928.

3. Cooke, J. V.: Scarlet Fever: V. The Changes in Skin Sensitivity to Scarlatinal Streptococcus Filtrate in Children Following the Introduction of Homologous Serum, Am. J. Dis. Child. **35**:784 (May) 1928.

The routine skin tests, as before, were carried out with 2, 20 and 50 standard skin test doses (STD) of scarlatinal filtrate toxin. These amounts were arbitrarily chosen and considered to represent a small quantity, a moderate quantity and a large quantity of toxin, respectively. The toxin employed in the skin tests was the same as that used previously [2] and contained 100,000 STD per cubic centimeter. For the injections of toxin, which were always given intramuscularly in volumes of 0.5 or 1 cc., dilutions of this same toxin preparation were used in some instances, and in others a "polyvalent" filtrate toxin was employed. The latter was made by mixing the broth filtrates from six strains of scarlatinal streptococci so diluted that each cubic centimeter of the filtrate, before mixing, contained 35,000 STD. The combined filtrates or "polyvalent toxin" showed this same titer. Blood was not used in preparing either of these toxins, which were furnished through the kindness of Mr. F. G. Jones of the Eli Lilly Biological Research Laboratories.

The skin reactions were read in twenty-four hours, and only those which reached a diameter of 10 mm. were regarded as positive. It was relatively rare, however, that any redness of the skin occurred when reactions were considered negative, although an occasional rather small area of erythema, from 3 to 6 mm., could be seen. One type of positive reaction to which special attention will be called is a much modified or "ghost" reaction. In this, the area of erythema which is of the same size as the usual positive reaction, is pale and faint. When this kind of reaction was noted, a larger amount of toxin usually gave the more intense redness of the ordinary positive reaction.

Only the cases of those persons who had well marked typical reactions, 15 mm. or larger to 2 STD of toxin, and no reaction in a heated control test, were studied. All were clinically free from any infection. Most of the children were patients at the Shriner's Hospital for Crippled Children, who were being treated for orthopedic deformities; they were studied by permission of Dr. Leroy Abbott.

A preliminary series of tests may be mentioned briefly.

1. A child with sensitive skin, aged 6, without neutralizing antibody in the blood, was given doses of 250 and 500 STD intramuscularly at an interval of four hours. Skin tests made the following day showed that sensitivity to 2 STD of toxin had been completely lost, although 20 STD still caused a positive reaction. On the fourth day after the injections, however, the reaction was again positive to 2 STD as it was on the seventh and tenth days. Antitoxin was not found on the fourth and eleventh days.

2. A boy, aged 13, showing the same sensitivity of the skin and lack of antitoxin, was given the same amounts of toxin and in addition 1,000 STD six hours later. In this instance, the loss of skin sensitivity to the

small amount (2 STD) of toxin was noted in the skin reactions, not only on the day following, but also in four successive tests (fourth, seventh, tenth and fifteenth days) during the next two weeks, although 20 STD continued to cause positive reactions. On and after the twenty-first day, sensitivity to 2 STD was again present. Antitoxin was not found on the fourth, eleventh or twenty-first days.

In neither of these children was there any local or general reaction following injections of toxin.

3. Each of a group of five young adults (nurses) with marked sensitivity of the skin to 2 STD of toxin and without antitoxin in the blood, received 250, 500 and 1,000 STD of toxin at four-hour intervals. In all, some general reaction followed with slight fever and malaise and local soreness and swelling of the arm at the point of injection. This reaction was similar to that observed after vaccines and diphtheria toxin-antitoxin mixtures had been used for adults. In none was there any generalized erythema of the skin or other sign of specific scarlatinal toxemia. Following the injections of toxin, the skin sensitivity to 2 STD of toxin had disappeared in tests made the next day in only one, and this negative reaction persisted for as long as five weeks, although the reactions to 20 STD continued to be positive. In three the skin reactions to 2 STD, which had been well marked and typical, had become much modified the day following and continued so during the following five weeks. The reactions with 20 STD, however, remained as well marked as they had been previous to the injections. In the other subject, a change in the skin reactions did not occur. None developed demonstrable antitoxin during the period of observation.

In these preliminary tests, it seemed that a definite change in skin sensitivity to filtrate toxin followed rather quickly after the injections of toxin. The severity of the local reaction following the injections of even 0.01 cc. of the filtrate in adults made it advisable to continue the observations in children.

The first series consisted of eight children from 5 to 13 years of age, who were given a polyvalent toxin with a titer of 35,000 STD per cubic centimeter. All were given 350 STD (0.01 cc.) with 700 STD (0.02 cc.) five hours later, and 1,400 STD (0.04 cc.) the following day. Four were given an additional 3,500 STD the third day. Skin tests were made every second day during the first ten days, and at least once weekly for a number of weeks. One child could be followed for only two weeks and another for only three weeks. The blood was tested for antitoxin at the beginning of the third, fourth and sixth weeks after the injections. The reactions following the injections of toxin were mild locally without general malaise or fever. In the four children who received approximately 6,000 STD during three days, a generalized

scarlatiniform erythema of a mild degree was noted on the fourth and fifth days, but other toxic symptoms were not present. The skin reactions of these children who developed a rash did not show essential differences from those who did not.

The changes in skin sensitivity following the injections of toxin in these children are shown in chart 1. In this is shown the smallest amount of filtrate to which a positive reaction was given on the days following the injections. The usual positive reactions and the modified, faint "ghost" reactions are indicated. It will be seen that on the second day, two had lost the skin sensitivity to small doses of toxin and in four others, the reaction was much modified; on the fourth day only four gave positive reactions to 2 STD, and three of these were "ghost" reactions. After this the skin sensitivity to a small quantity of toxin was lost in all but one, although when a moderate quantity (20 STD) was given, the reactions in all except two children continued to be positive. After ten weeks there was some evidence of an increase in skin sensitivity

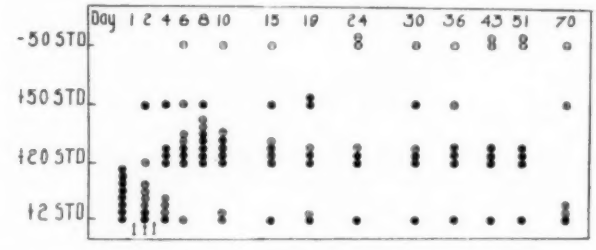

Chart 1.—Changes in skin sensitivity following injections of small amounts of scarlatinal streptococcus filtrate toxin. In this and in chart 2 the line within the circle indicates negative reaction; the cross within the circle, a modified "ghost" reaction; the solid circle, the usual positive reaction and the arrows indicate injections of toxin.

in two of the children. In the children in this series a definite and rather constant decrease in skin sensitivity to scarlatinal toxin occurred soon after intramuscular injections of the same streptococcus filtrate.

The relation of this decrease in skin sensitivity to the presence of neutralizing antitoxin in the blood of these children is shown in table 1. In only two was antitoxin demonstrable, one early in the third week and the other at the beginning of the fourth week after the first injection of toxin. The loss of skin sensitivity in these two was the most marked and prolonged of any in the group (chart 1). Antitoxin could not be found in four others followed for six weeks. The absence of skin sensitivity to a small quantity of toxin after such injections apparently was not always accompanied by the presence of demonstrable circulating antitoxin, and certain children who later became immune had a marked

decrease in skin sensitivity from one to two weeks before circulating antitoxin was demonstrable.

The case of one child (no. 3 in table 1) is worthy of brief comment. He was of the group given a total of 6,000 STD during three days, and he developed a mild scarlatiniform rash on the next two days. The following day the skin reaction to 2 STD of toxin was negative, although the reaction to 20 STD was still positive. This degree of sensitivity persisted for seven weeks, after which the skin again reacted to 2 STD, for the three succeeding weeks, during which tests were made. Antitoxin was not found by tests made during five weeks after the injections. Three months after the last skin tests, this child developed typical clinical scarlet fever. He had been in bed in an orthopedic hospital ward and had not been in contact with any one who had an acute infection.

TABLE 1.—*Development of Neutralizing Antitoxin in Blood after Smaller Injections of Toxin (First Series)*

		Tests for Antitoxin			
			Day After First Injection		
Child Number	Age	Before Injections	15th	21st	37th
1	8	—	+	+	+
2	5	—	—	+	+
3	5	—	—	—	—
4	6	—	—	—	—
5	7	—	—	—	—
6	13	—	—	—	—
7	5	—	—		
8	10	—	—		

The second series consisted of seven children, from 7 to 15 years of age, who were given much larger amounts of the same filtrate, and the skin sensitivity and development of antitoxin followed in the same manner. Each was given the following amounts of a polyvalent filtrate toxin: first day, 350 STD (0.01 cc.) and five hours later 700 STD (0.02 cc.); third day, 1,400 STD (0.04 cc.); fifth day, 3,500 STD (0.1 cc.); seventh day, 8,750 STD (0.25 cc.), and ninth day, 17,500 STD (0.5 cc.). A final dose of 35,000 STD (1 cc.) was given to three of the children on the eleventh day. Only one child gave marked local reactions from the injections, and in one other a rise in temperature to 103 F. followed several of the injections, but other symptoms were not present. None had a rash.

The changes in the skin sensitivity to varying amounts of toxin are given in chart 2. They were of the same character as in the previous series (chart 1) but were more marked. The day following the first injections, three children gave negative reactions to 2 STD of toxin, and three others gave only faint, modified reactions to this amount. By the eighth day, all had lost the skin sensitivity to a small quantity (2 STD)

of filtrate toxin, and by the tenth day half of them gave negative reactions to a large quantity (50 STD). After the thirtieth day, there was a relative return of skin sensitivity, since some who had previously given a negative reaction to the larger doses, gave positive reactions with 20 STD. By the tenth week, two again gave weakly positive reactions with 2 STD of toxin. In this series of children given larger amounts of toxin (30,000 to 67,000 STD), the decrease in skin sensitivity appeared quickly and became progressively more marked. After some weeks, there was a definite return of skin sensitivity, although the marked degree of skin sensitivity present before the injections did not return during the period of observation (ten weeks).

The relation of this loss of skin sensitivity to the development of circulating neutralizing antibody in this series was also studied, and the results of the determinations for antitoxin are shown in table 2. As

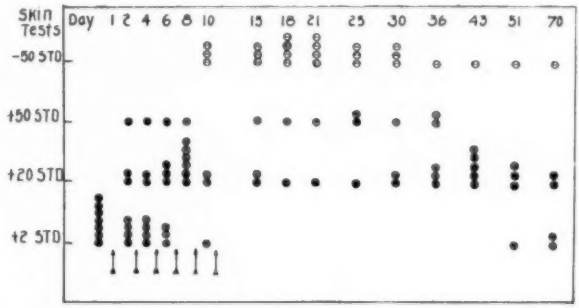

Chart 2.—Changes in skin sensitivity following injections of large amounts of scarlatinal streptococcus filtrate toxin.

indicated, a number of these determinations were verified by blanching tests with 0.5 cc. of serum on early scarlet fever rashes. One child was found to have neutralizing antitoxin before the injections were given. The original skin sensitivity to toxin was apparently as marked in this subject as in the others, and the changes in sensitivity after the injections were apparently identical with those of the others in this group. It appears that the association of demonstrable antitoxin with an absence of skin sensitivity to small quantities of toxin, while common, is not absolutely constant. Three other children had demonstrable antitoxin during and after the third week, and another early in the fourth week. In each of these four, the skin sensitivity had been greatly decreased for a number of days before antitoxin appeared in the blood. One child did not show antitoxin for at least seven weeks, although the decrease in skin sensitivity to toxin was as marked as the others in whom antitoxin developed. This child was one in whom skin sensitivity to a

small quantity of toxin returned after a number of weeks. In another who could be followed for only two weeks, antitoxin was not found. During the period of administration of toxin, the serum of five of the children was tested for toxin by intracutaneous injections into other children who were sensitive to 1 STD or less. In none did 0.3 cc. of serum give any reaction (table 2).

COMMENT

While studies of the Dick test during the first week after injections of toxin have not been reported, some observers (Dick and Dick,[1] Larsen and others,[4] Colby,[5] Lees [6]) have noted that the reaction became negative as early as the sixth to the eighth day after a single injection. Negative reactions to the Dick test after injections of toxin have, in general, been

TABLE 2.—*Development of Neutralizing Antitoxin in Blood After Larger Injections of Toxin (Second Series)*

		Tests for Antitoxin						
		Before Injections	Day After First Injection					
Child Number	Age		5th	11th	17th	22d	31st	43d
1	13	+*	+†	+†	+*	+	+	+
2	11	—	—‡	—	+	+	+	+
3	9	—	—†	—*	+*	+	+	+
4	15	—	..	—	+	+	+	
5	12	—*	..	—	—	+	+	
6	7	—	—†	—†	—*	—*	—	—*
7	12	—*	—†	—†				

* Result was verified by blanching test with 0.5 cc. of serum.
† Result was verified by blanching test with 0.5 cc. of serum, and tests for circulating toxin were negative with 0.3 cc. of serum.
‡ Tests for circulating toxin were negative using 0.3 cc. of serum.

assumed to indicate immunity. The decrease in skin sensitivity which followed injections of scarlatinal streptococcus filtrate toxin in the children here considered occurred in many instances too quickly to be explained by the development of a specific immunity. Usually, the production of demonstrable antibodies following artificial immunization requires a certain number of days. In the children studied, demonstrable neutralizing antibody was not recognized until at least a week after a definite and often striking decrease in skin sensitivity had been observed. The rather rapid decrease in sensitivity of the skin suggests a process similar to the desensitization of anaphylactically hypersensitive animals

4. Larsen, W. P.: Huenekens, E. J., and Colby, W.: Immunization Against Scarlet Fever with Toxin Detoxified with Sodium Ricinoleate, J. A. M. A. **86**:1000 (April 3) 1926.

5. Colby, W.: The Control of Scarlet Fever in Institutions, J. A. M. A. **87**:919 (Sept. 18) 1926.

6. Lees, H. D.: The Dick Test, with Active and Passive Immunization for Scarlet Fever, J. A. M. A. **88**:1133 (April 9) 1927.

by small but increasing injections of antigen. The well known experiments of Besredka have shown that the injection into sensitized guinea-pigs of amounts of antigen considerably below the lethal dose is followed by a period during which the animal is protected from several times the lethal dose. The desensitization is relative, and in order to increase the tolerance of the animal to large doses of the antigen, it is necessary to give increasing amounts of the antigen at short intervals. In other words, animals may be partially or completely desensitized, depending on the number of preliminary injections of antigen. The desensitization is temporary, and sensitivity returns after a varying number of days.

Between the phenomenon of desensitization to proteins in anaphylactic animals and the desensitization of the human skin to scarlatinal streptococcus filtrate, there are, however, certain differences which relate to the speed of the two processes. In the desensitization of animals, the change in reactivity of the body cells caused by the action of subshocking doses of antigen is rather abrupt, and the loss of sensitivity to large amounts of antigen is demonstrable almost immediately afterward. There is also a sudden loss of hypersensitivity after even mild anaphylactic reactions in animals. In cutaneous desensitization to the scarlatinal streptococcus filtrate in man, on the other hand, while a decrease in sensitivity may occur soon after injections of antigen, it is only after several days that this decrease usually becomes striking. Even when the antigen was given sufficiently rapidly, or in large enough doses to produce a generalized cutaneous scarlatiniform reaction, a sudden loss of skin sensitivity did not follow. If the loss of sensitivity following the injections of scarlatinal streptococcus filtrate in man is to be considered analogous to anaphylactic desensitization to proteins in animals, one must assume a slower union of antigen with cellular antibodies in the human reactions. It is of interest, in this connection, that those infants in whom skin sensitivity to filtrate toxin developed after the injection of homologous serum [3] showed positive skin reactions only after several days.

In spite of the greater rapidity of the phenomenon of desensitization in animals, the early loss of cutaneous sensitivity to scarlatinal streptococcus filtrate resembles this process more closely than the development of skin immunity to a soluble toxin such as diphtheria, which is dependent entirely on the formation of a specific antitoxin and requires a number of days or weeks. The more rapid loss of skin sensitivity to scarlatinal streptococcus filtrate after injections seems distinct from such immunity, although the later production of neutralizing antibody indicates that a true antibody immunity may prevent the return of skin sensitivity for a considerable period.

SUMMARY

The injection of scarlatinal streptococcus filtrate toxin in increasing amounts at short intervals caused a relatively rapid and, in several cases, a marked decrease in skin sensitivity to the toxin. In over one third of the persons studied, the reaction to the Dick test, or skin sensitivity to a small quantity of toxin, had disappeared twenty-four hours after the injections of toxin, and in most cases the skin sensitivity was much modified after this short interval. Children given larger amounts of the filtrate toxin showed a greater decrease in skin sensitivity, and some gave negative skin reaction to a considerable quantity (50 STD) of toxin during the second week.

Neutralizing antitoxin was not found in the blood coincidently with the decrease in or loss of skin sensitivity, but was demonstrable only in the third or fourth week after the injections were started. In some, even those who showed a marked decrease in sensitivity, antitoxin could not be recognized. When antitoxin appeared, it continued to be demonstrable, at least for some weeks.

After a few weeks there was a tendency for the decrease in skin sensitivity to be less marked, and in some later tests, weakly positive reactions to the Dick tests reappeared.

To explain this relatively rapid loss in skin sensitivity and the slower development of neutralizing antibody following the injections of scarlatinal streptococcus filtrate toxin, the resemblance to certain anaphylactic phenomena in animals was pointed out. The possibility was suggested that there occurs, first, a desensitization of body cells anaphylactically sensitive to the scarlatinal streptococcus filtrate and, somewhat later with continued absorption of antigen, a development of specific antibodies.

SCARLET FEVER

VII. THE COURSE OF THE DEVELOPMENT OF IMMUNITY IN SCARLET FEVER*

JEAN V. COOKE, M.D.

ST. LOUIS

In a study [1] of the changes in skin sensitivity to scarlatinal streptococcus filtrate toxin, and the development of neutralizing antitoxin following injections of the filtrate, it was found that the decrease in skin sensitivity appeared relatively quickly, and that antitoxin was not usually demonstrable until a week or two later. The rapid change in skin sensitivity resembled desensitization of an anaphylactically hypersensitive tissue following injection of antigen, and appeared not to be dependent on the later formation of specific antibodies. In the present paper, observations on the changes in skin sensitivity and the development of antitoxin in the course of clinical scarlet fever are reported.

The reaction to the Dick test in scarlet fever, according to a number of reports, is modified by the disease and tends to become negative during convalescence. Before the onset, the reaction has been positive in almost all those tested, and, in many instances, during the first week of the infection, although there is some variation in the proportion of cases reported with positive reactions to the Dick tests during the exanthem. It is mentioned by several observers that it was frequently difficult to determine positive reactions in the course of the rash, and Dick [2] says that the reaction may become negative during the first forty-eight hours of the disease.

In order to illustrate the loss of skin sensitivity during convalescence, as well as to show the variations reported during the first week, the figures of several observers are collected in table 1. It will be seen that although some report the reaction to the Dick test to be constantly positive during the first week, others found many with negative reactions during this period. It may be that such discrepancies resulted from the difficulties of interpreting reactions on an exanthematous skin, or from the fact that tests made early may differ from those made toward the end of the first week. A large number of the positive reactions that occurred early in the disease are termed "faintly positive." In all

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* Received for publication, Jan. 9, 1928.

* From the Department of Pediatrics, Washington University School of Medicine and the St. Louis Children's Hospital.

1. Cooke, J. V.: Scarlet Fever: VI. The Relation Between the Loss of Skin Sensitivity to Toxin and the Development of Antitoxic Immunity During Artificial Immunization, Am. J. Dis. Child., this issue, p. 974.

2. Dick, G. F.: Methods for the Control of Scarlet Fever, J. Michigan M. Soc. **26**:351 (June) 1927.

reports only a small proportion of positive reactions to the Dick test was found during convalescence.

Of interest in this connection are the reports of those who have observed the changes in skin sensitivity by more frequent Dick tests during the disease. In order to illustrate the rather striking decrease in skin sensitivity early in the infection, the results of five different series of tests are shown in the chart.

The figures shown are from Ker, McCartney and McGarrity,[3] Rosen and Korobicina,[4] Silcock,[5] Joe,[6] and Smith and Taylor[7] and include 603 tests during the first week and 376 during the second week. In the chart, the percentage indicated on a given day of the disease includes all tests on the days since the preceding charted percentage. Four of the five curves show an increasing and rather rapid loss in

TABLE 1.—*Reactions to the Dick Test During the First Week of Scarlet Fever and During Convalescence*

Observer	First Week		Convalescence	
	Number	Per Cent Positive	Number	Per Cent Positive
Branch and Edwards (J. A. M. A. **82:** 1260 [April 19] 1924)..	3	100	65	0
Zingher (J. A. M. A. **83:** 432 [Aug. 9] 1924).....	170	100	170	0
Gorter and others (Acta Paediat. **6:** 383, 1927).....	30	100	30	7
Meyer (Ztschr. f. Kinderh. **43:** 258, 1927).....	142	82.5	142	23.2
Harris and Macfarlane (Pub. Health, London, **39:** 216, 1926)	72	60	96	15.6
Lesné and Dreyfus-Sée (Bull. et mém. Soc. méd. d. hôp de Paris. **50:** 452, 1926).....	12	58	15	20
Debré and others (Bull. et mém. Soc. méd. d. hôp de Paris. **50:** 476, 1926).....	19	42	65	1.5
Krstulovic (Monatschr. f. Kinderh. **33:** 120, 1926).....	25	33	..	....
Brown (Brit. J. Child Dis. **22:** 171, 1925).....	30	26.6	60	10
Dick and Dick (J. A. M. A. **82:** 265 [Jan. 26] 1924).....	..	.....	65	4.6
Okell and Parish (Lancet. **208:** 712, 1925).....	..	.....	120	18

skin sensitivity during the first week during which the Dick reaction became negative in approximately half of the cases. The decrease in positive reactions to the Dick tests later is gradual during convalescence, and a small proportion continued to be positive. In one series (Joe) the results varied considerably from the other figures, and the decrease in positive reaction to the Dick tests did not occur until later in convalescence. This discrepancy is probably due chiefly to Joe's method of

3. Ker, C. B.; McCartney, J. E., and McGarrity, J.: The Dick Test for Susceptibility to Scarlet Fever, Lancet **208**:230 (Feb. 7) 1925.

4. Rosen, P. S., and Korobicina, L. A.: The Scarlet Fever Toxin and the Dick Test, J. A. M. A. **84**:1476 (May 16) 1925.

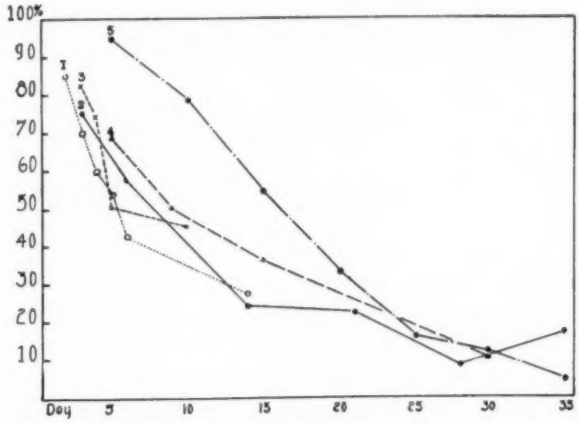
5. Silcock, F. A. E.: The Dick Test at a Fever Hospital, Lancet **209**:1324 (Dec. 26) 1925.

6. Joe, A.: A Clinical Study of the Dick Test, Lancet **209**:1321 (Dec. 26) 1925.

7. Smith, J., and Taylor, J. S.: The Dick Test in Scarlet Fever Patients and in Normal Individuals, J. Hyg. **25**:90, 1926.

reading the tests. He not only included as positive those smaller reactions "from one-half to one-fourth the usual size" which were called doubtful or negative by others, but also called positive those in which some reaction appeared eight hours after the test, although it was negative at twenty-four hours. Aside from the question of the validity of his standard for a positive reaction, this marked variation between his methods for reading tests and that of other workers, makes it difficult to compare his results with theirs. Excluding Joe's series, there is noted a constant loss in skin sensitivity to toxin during the first week of the disease in a large number of cases of scarlet fever.

The development of antitoxin after the fading of the rash has been studied in eleven instances by Blake and Trask,[8] who found no antitoxin in six patients between the seventh and thirteenth days of the



Positive reaction to Dick tests during scarlet fever as given by various observers. Curve 1 shows the results of Smith and Taylor in 170 cases; 2, those of Ker and others in 400 cases; 3, those of Rosen and Korobicina in 103 cases; 4, those of Silcock in 155 cases and 5, those of Joe in 681 cases.

disease. In all these a small amount of toxin was still demonstrable in the blood. Three others showed antitoxin (two on the fourteenth day and one on the nineteenth day), and two had no antitoxin on the twenty-first day. These results indicate that the disappearance of the rash is not due to the development of antitoxin.

MATERIAL AND METHODS

Eleven cases of scarlet fever from the St. Louis Children's Hospital and thirteen from the St. Louis City Isolation Hospital were studied.

8. Blake, F. S., and Trask, J. D.: Studies in Scarlet Fever: II. The Relation of the Specific Toxemia of Scarlet Fever to the Course of the Disease, J. Clin. Investigation **3**:397 (Dec. 20) 1926.

The latter were followed through the kindness of the superintendent, Dr. Curtis Lohr, who assisted with the tests. Eight of the patients were adults, and seven of the children were under 6 years of age. All cases were typical clinically, but none of the patients had severe intoxication. Only two had complications, one a simple otitis, and the other, otitis, hemorrhagic nephritis, mastoiditis and fatal hemolytic streptococcus septicemia.

Skin tests were made with 2, 20 and 50 standard skin test doses (STD) of a potent toxin as previously described,[9] at least once weekly and in some cases every fourth or fifth day. They were read in twenty-four hours. The reliability of the toxin was being constantly controlled by a number of tests on normal children. Neutralization tests for antitoxin were performed in most cases once a week. In thirteen cases, tests for toxin in the circulating blood were carried out as described by Blake and Trask [8] using 0.3 cc. of serum. Each serum was tried on from two to five subjects whose reactions to the Dick tests were positive. Many of the positive reactions to serum toxin were faint but readily measurable.

Changes in Skin Sensitivity.—In three children, skin reactions had been positive to 2 STD a short time before the onset of scarlet fever. The results of the tests made during the course of the disease are shown in table 2. When more than one test on a patient was made during the seven day period shown, only the stronger one was tabulated. Most of the cases could be studied for only three weeks, but some were followed for five weeks. During the first week of the disease, it will be seen that a relative loss in skin sensitivity occurred in a number of instances, if one can assume that all had as marked a skin sensitivity before the onset as did the three who were tested. The proportion giving positive skin reactions to a small amount (2 STD) of toxin is lower than that reported by most others, and may be due to the small series studied. Some reactions to the tests made during the period of the rash were difficult to interpret with certainty, especially those when the smallest quantity (2 STD) of toxin was used. To be certain always whether or not there was a slight intensification of the erythema at the area of the test was almost impossible. For this reason, only those were called positive in which the erythema was sufficiently increased at the site of the test as to be unmistakable. As a consequence, it may be that some of the fainter reactions were missed. It seems likely, however, that coincident tests with the larger amounts of toxin would have been readily seen unless a marked change in the skin sensitivity had occurred.

9. Cooke, J. V.: Scarlet Fever: I. The Relation Between Antitoxin in the Blood and Skin Sensitivity to Toxin in New-Born Infants and their Mothers, Am. J. Dis. Child. **34**:969 (Dec.) 1927.

When the rash was absent at the site of the skin test, difficulty in reading the reactions was not encountered.

It will be seen (table 2) that during the first week some patients required 50 STD of toxin to give a positive skin reaction and others gave a negative reaction even to this amount. During the second and third weeks, the loss of skin sensitivity to larger amounts of toxin was noted in a still larger number. The series studied is too small to warrant the tabulation of percentage changes in skin sensitivity. It is evident, however, not only that an early decrease in skin sensitivity to toxin occurs in many cases of scarlet fever, but that in a number of them the skin does not react to the injection of considerable amounts of toxin. This loss of skin sensitivity involves an increasing number of patients during the second and third weeks of the disease.

In three instances an increase in skin sensitivity was observed during convalescence. Two children gave negative skin reactions to 50 STD during the first and second weeks and a positive reaction to 20 STD

TABLE 2.—*Changes in Skin Sensitivity During Scarlet Fever*

Day of Disease	No.	Skin Reactions			
		+ 2 STD	+ 20 STD	+ 50 STD	— 50 STD
2-7	23	7	10	3	3
8-14	23	2	9	5	7
15-21	24	0	9	6	9
22-28	8	0	2	2	4
28-35	6	0	2	2	2

during the third week. One of these, when retested during the eighth week, gave a faintly positive reaction to 2 STD. The third child gave a negative reaction to 50 STD for four weeks, and during the fifth, sixth and seventh weeks gave positive reactions to 20 STD. It is of interest in this connection that Zingher [10] reported three children in whom the reaction to the Dick test which had been positive during the first week became negative during the second or third weeks, and again positive during the fifth week. In one of these antitoxin was not found on the thirty-second day. Apparently, the decrease in skin sensitivity which accompanies scarlet fever is merely temporary in certain cases.

Toxin in the Circulating Blood.—Thirteen cases were tested for toxin in the serum by injecting 0.3 cc. intradermally into children with a positive reaction to the Dick test. The results of such tests are shown in table 3 in which the state of the rash and the degree of skin sensitivity at the time of the toxin tests are also noted. Toxin

10. Zingher, A.: The Dick Test in Normal Persons and in Acute and Convalescent Cases of Scarlet Fever, J. A. M. A. **83**:432 (Aug. 9) 1924.

was demonstrable in all except one from the second to the eleventh days after the onset of symptoms. It was found during fully developed and fading rashes and after the eruption had disappeared. No relation to the degree of skin sensitivity was demonstrable since positive reactions to toxin were given by some who were sensitive to 2 STD and by others whose skin was not sensitive to larger amounts. As controls, the serums of eight children with a positive reaction to the Dick test

TABLE 3.—*Relation of Toxin in the Blood to Rash and Skin Sensitivity*

Case No.	Age	Day of Disease	Rash	Skin Test	Toxin
1	3	2	Moderate	+ 2 STD	+
2	5	2	Marked	+ 2 STD	+
3	7	3	Moderate	+ 20 STD	+
4	3	3	Moderate	+ 50 STD	+
5	5	5	Moderate	+ 50 STD	+
6	25	5	Marked	+ 20 STD	+
7	18	5	Moderate	+ 2 STD	+
8	24	5	Fading	+ 20 STD	+
9	27	5	Fading	+ 2 STD	+
10	18	5	Fading	+ 20 STD	+
11	7	6	Fading	— 50 STD	—
12	7	6	Fading	+ 2 STD	+
13	6	8	Gone	+ 20 STD	+
6	25	11	Gone	— 50 STD	+

TABLE 4.—*Development of Antitoxin in Scarlet Fever*

	Days of Disease on Which Tests Were Made	
Case No.	Antitoxin Absent	Antitoxin Present
1	2*	18
2	3	9, 16
3	5	10, 15, 26
4	5*	11, 21
5	5	11, 21
6	6	11, 16, 27
7	3	13, 33,
8	6*	13
9	5*, 10	16
10	5*, 11*	17
11	3*, 9	19
12	5*, 12	19
13	6, 14	20, 25, 34
14	5, 15	22, 32
15	3, 15	.....
16	4*, 15	.....
17	5*, 12, 19	.....
18	5*, 11, 21	.....
19	6*, 12, 22	.....
20	8*, 15, 22	.....

* Toxin present.

were similarly injected intradermally in 0.3 cc. amounts into other children with a positive Dick reaction. A reaction did not occur in any of these tests.

Development of Antitoxin.—In twenty cases, samples of blood were tested for antitoxin at intervals of five days or more during the first three weeks of the disease. The results of these tests are shown in table 4 in which are summarized sixty-one tests on the twenty patients. The earliest that antitoxin was demonstrated was the ninth day after

the onset of symptoms. It will be noted that in a number of cases antitoxin was found during the second week, while in others it did not appear until the third week. In some, antitoxin was not demonstrable even after three weeks. It is of interest that when antitoxin once appeared in the blood, it was constantly demonstrable later. Apparently, the time of development of antitoxin in scarlet fever varies in different persons and commonly appears during the second or third week, although in some cases its development is delayed until later. Such variations may possibly be associated with differences in the amount or rapidity of the absorption of antigen which must occur, and on individual differences in antibody response. Antitoxin was not demonstrable until some days after the rash had disappeared.

Quantitative estimation of the amount of neutralizing antitoxin was not attempted in the cases tabulated. That a marked difference exists in the amount of antitoxin in different persons during convalescence, however, is indicated by some observations made on other cases. In seven children in whom blood was obtained between the twenty-eighth and thirty-second day after the rash was first noted, antitoxin was found in the undiluted serum but not in 1:10 dilutions in two; in 1:10 but not in 1:25 in one; in 1:25 but not in 1:50 in three; and in 1:100 dilution in one.

SUMMARY

The changes in skin reactivity and in the neutralizing power of the blood for toxin which take place in scarlet fever may be summarized briefly as follows. The skin, which has a marked sensitivity to a small quantity of toxin before the onset of the disease, tends to become less sensitive as the eruption progresses. In most cases definite decrease in sensitivity occurs quickly as the rash appears; in many, the reaction to a small quantity is modified and faint; in others, a reaction to considerable amounts of toxin does not occur; and in a few the sensitivity is unaltered. By the end of the first week the skin sensitivity has become much decreased, although some still react to a small quantity of toxin. Later, the decrease in skin sensitivity becomes more marked and more constant. In a few there is a late return of skin sensitivity. During the eruption, toxin is demonstrable in the circulating blood in many cases, and even after the rash has disappeared, it may still be found. Neutralizing antitoxin does not appear until the second or third week, or even later.

Certain of these phenomena are not readily explained on the hypothesis that the scarlatinal toxin is a primarily toxic substance. For example, if the skin rash results from the action of a true toxin, one would not expect free toxin to be present in the circulation while the rash is fading or after it has disappeared. Also, the lack of demon-

strable antitoxin in the blood until some days or weeks after the eruption has faded indicates that the disappearance of the rash is not related to the development of antitoxin.

The decrease in skin sensitivity to toxin and the development of antitoxin noted in scarlet fever are similar to the changes which were observed to follow the repeated injection of toxin in children with positive reactions to the Dick test.[1] In both, the change in skin reactivity was noted relatively early. The development of neutralizing antitoxin in the blood appeared later, and in some instances there was a late increase in skin sensitivity. It would seem justifiable to conclude that under both conditions the changes are responses to the absorption of the scarlatinal toxin and that any quantitative differences may be due to the variation in the rapidity and amount of toxin absorbed.

The generalized reaction of the skin to the toxin in scarlet fever has certain characteristics which suggest a reactive ability of the cells which become exhausted. Typically, the rash increases during the first two or three days to its maximum and then requires about two days to fade. During this time and even after the disappearance of the rash, the toxin continues to be demonstrable in the blood. This leads to the conclusion that the cellular reactivity has become exhausted with inability to respond further to the action of the toxin. Such a phenomenon is similar to the saturation of antibodies of sensitized cells by circulating antigen and is apparently distinct from the action of primarily toxic substances. The effect of immune serum on this cellular reaction seems to harmonize with the hypothesis of a limited reactive ability of the cells. The local (Shultz-Charlton) and general (therapeutic) effects of immune serum are observable only when the serum is used early. Apparently, until the rash has reached its height, due to the maximum effect of the toxin on the cells, immune serum has the ability to inhibit further cellular reaction by its greater avidity for the toxin, and that already present recedes in a short time. After the cellular response to toxin has reached its fastigium, however, the immune serum has no effect, since the cellular reactive ability to toxin has become exhausted.

The action of toxin in scarlet fever resembles more the desensitization of cells which are anaphylactically hypersensitive than the effect produced by a true soluble toxin.

SCARLET FEVER

VIII. THE ANAPHYLACTIC FACTOR IN SCARLET FEVER*

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In a series of studies[1] on the sensitivity of the skin to scarlatinal streptococcus filtrate toxin and its neutralizing antibody, the following facts have been observed:

1. In the new-born, the skin was usually not sensitive to considerable amounts of scarlatinal toxin, and this lack of sensitivity was not due to the presence of antitoxin in the blood. Relatively few infants developed sensitivity during the early months of life, but by the end of the first year the skin had become sensitive in a large proportion. Although antitoxin was not demonstrable in many young infants whose skin was insensitive to toxin, in older children the lack of sensitivity was practically always accompanied by antitoxin in the blood.

2. Hemolytic streptococci isolated from young infants with infections of the upper respiratory tract in certain instances formed a toxin which was neutralized by convalescent scarlet fever serum, but not by serum from children whose skin was sensitive to scarlatinal toxin. The skin of a high percentage of infants and young children in an institution in which hemolytic streptococcus infection of the upper respiratory tract was prevalent was not sensitive to the toxin.

3. During measles there was a temporary loss or decrease of skin sensitivity to toxin similar to that which occurs to tuberculin and horse serum during this disease.

4. Certain infants whose skin was not sensitive to the toxin became so following an injection of serum from a sensitive person. The injec-

* Received for publication, Jan. 9, 1928.

* From the Department of Pediatrics, Washington University School of Medicine and the St. Louis Children's Hospital.

1. Cooke, J. V.: Scarlet Fever: 1. The Relation Between Antitoxin in the Blood and Skin Sensitivity to Toxin in New-Born Infants and Their Mothers, Am. J. Dis. Child. **34**:969 (Dec.) 1927. II. The Development of Toxin Sensitivity of the Skin in Infants and Its Relation to the Presence of Antitoxin in the Blood, ibid. **35**:762 (May) 1928. III. Modification of Skin Sensitivity to Toxin as a Result of Nonscarlatinal Infections, ibid. **35**:772 (May) 1928. IV. The Toxin of Hemolytic Streptococci from Nonscarlatinal Infections in Infants, ibid. **35**:781 (May) 1928. V. The Changes in Skin Sensitivity to Scarlatinal Streptococcus Filtrate Toxin in Children Following the Introduction of Homologous Serums, ibid. **35**:784 (May) 1928. VI. The Relation Between the Loss of Skin Sensitivity to Toxin and the Development of Antitoxic Immunity During Artificial Immunization, ibid., this issue, p. 974. VII. The Course of the Development of Immunity in Scarlet Fever, ibid., this issue, p. 983.

tion of serum containing neutralizing antitoxin into children with a sensitive skin was followed by an immediate decrease in skin sensitivity.

5. Following injections of increasing amounts of toxin at daily or two day intervals, sensitive children showed a relatively rapid decrease or loss in skin sensitivity. Neutralizing antitoxin, however, did not appear for a number of days or weeks.

6. In clinical scarlet fever, a similar decrease in the sensitivity of the skin to toxin occurred during the first week of the disease. The toxin could be demonstrated in the circulating blood after the fading of the rash with marked decrease in skin sensitivity. Neutralizing antitoxin was not found until the second or third week.

Before commenting on the interpretation of the foregoing results, it is of interest to review briefly the evidence of the production of toxin by scarlatinal streptococci, and to compare the action of scarlatinal toxin with other well known exotoxins such as diphtheria toxin. Between these two there are certain analogies. The essential features of streptococcus filtrates on which the assumption of toxin production is based are well known. They are (1) the production of a local erythematous reaction (Dick reaction) in the skin of certain human beings after the intradermal injection of a small amount of the filtrate; (2) the presence in the blood of convalescent scarlet fever patients, many persons with negative reactions to the Dick test, and filtrate-immunized animals, of a substance which will inhibit the erythema-producing action of the filtrate when injected into susceptible skin; (3) the production of toxic symptoms such as fever, general malaise, vomiting and generalized skin rash by the injection of moderate amounts (1,000 STD) of filtrate into susceptible human beings; (4) the local blanching (Schultz-Charlton phenomenon) of early scarlatinal rashes by intradermal injection of convalescent or immune serum, and (5) the frequent beneficial clinical effect on the toxic symptoms and rash of scarlet fever by the injection of convalescent or immune serum when given relatively early. Some of these phenomena are strikingly similar to those noted with diphtheria toxin, and have led to a wide acceptance, especially in America, of the production of a true exotoxin by the scarlatinal streptococcus.

There are, however, other respects in which the scarlatinal streptococcus filtrates differ markedly from diphtheria and other bacterial toxins, and which are difficult to harmonize with the conception of true toxins. Some of these are as follows:

1. The thermostability of the streptococcus filtrate toxin; this requires heating from twenty minutes to an hour or more at 100 C. to be destroyed. One of the most constant characteristics of bacterial toxins, such as diphtheria, tetanus and botulinus, as well as similar phytotoxins, like ricin, abrin and crotin, is thermolability, since all known exotoxins are destroyed by a few minutes' heating to a temperature of

from 75 to 80 C. By analogy, the degree of heat required to destroy the skin-reacting quality of streptococcus filtrate suggests denaturing or destruction of biologic specificity of a protein by heat rather than the inactivation of a thermolabile toxin.

2. The relative lack of deterioration of the scarlatinal toxic filtrate with age. Dilutions of the toxin containing 1 or 2 STD to 0.1 cc. will continue potent in skin tests for weeks, and I have tested concentrated filtrates after two years and have not found a change in titer. The rapid deterioration of diphtheria toxin is well known.

3. The limitation of the toxic effect to human beings. The filtrate is innocuous and causes no toxic symptoms in other species even when given in relatively large amounts, if the volume of broth injected is kept below the amount which gives nonspecific toxic effects with bacterial filtrates in general. Rabbits, for example, have been given intravenously 500,000 STD, or approximately 500 times the amount which causes general toxic symptoms in susceptible persons, without showing any toxic effect. Guinea-pigs, dogs and other animals likewise are unaffected by the toxin. This immunity is not due to the presence of antitoxin in such animals, since neutralizing substances cannot be demonstrated in their blood. No such species specificity occurs with other known soluble toxins.

4. The character of the cutaneous reaction, the time of its appearance, the course and duration are dissimilar in the Schick test and in the Dick test.

5. A marked absence of demonstrable toxicity in the scarlatinal streptococcus filtrate during the early months of life. The only available method of testing toxicity is by intradermal injection, and the absence of skin toxicity of the filtrate, independent of the presence of neutralizing antitoxin in the blood in these young persons, suggests that even the species specificity of the filtrate toxin is an acquired characteristic, or one which is slow in developing. Nonspecific immunity to a true toxin is difficult to explain.

Other somewhat less striking discrepancies between scarlatinal filtrate and true toxins might be cited, but the foregoing ones appear sufficient to justify some skepticism in accepting the scarlatinal filtrate as an exotoxin. It is true that the exotoxins induce the production of specific neutralizing antibodies in the blood of a treated animal; but the fact that scarlatinal immune serum possesses the property of inhibiting or neutralizing the action of the streptococcus filtrate cannot, of itself, constitute proof that the filtrate contains toxin when the presence of a primarily toxic substance in the filtrate is otherwise inconclusive.

The difficulty in harmonizing certain facts already outlined with the action of a primarily toxic substance, as well as certain discrepancies between clinical observations on the sensitivity of the skin and the toxin

theory, prompts the examination of another hypothesis to explain the observed phenomena. The theory that certain of the symptoms of scarlet fever, including the skin rash, are dependent on a bacterial anaphylactic hypersensitivity has been advanced [2] from time to time, mainly on clinical grounds, and similarities have been pointed out between some of the clinical phenomena of scarlet fever, experimental anaphylaxis, and the human anaphylactic reaction in serum sickness. Among the striking clinical analogies between scarlet fever and serum sickness may be mentioned the sudden onset without prodromal signs, moderate, generalized swelling of the lymph nodes, sweating, fever, eosinophilia, arthritis of a similar type and a general exanthem. The latter symptom, while not identical in the two conditions, is often similar.

The antigen is presumably absorbed from the pharyngeal portal of entry in the usual form of scarlet fever and from the uterus or skin in the extrapharyngeal forms, such as the puerperal and surgical types and that following burns. Sensitization of susceptible persons is assumed to be acquired from repeated infections by streptococci, so that a later infection, although it may produce an insignificant local pharyngitis, causes the anaphylactic response. The variation in severity of the specific anaphylactic response is attributed to the degree of hypersensitivity in different persons, and the variability in the time of the appearance of the allergic symptoms after the onset of the infection, to the rapidity of formation and absorption of the antigen. The clinical immunity possessed by many persons, however, has been difficult to explain on the basis of lack of sensitization, since, in the widespread prevalence of hemolytic streptococcus infections, it would seem that few could escape such infection and consequent sensitization.

The discovery that filtrates of hemolytic streptococci from scarlet fever contain a skin-reactive substance furnishes, presumably, the hypothetic antigen and gives an opportunity to test experimentally the hypersensitivity under various conditions. Certain respects, such as greater thermostability and limitation of toxicity to man, by which the filtrate suggests a protein antigen rather than a toxin, have been mentioned previously. The relative absence of sensitivity of the skin in early infancy and the freedom of such young infants from clinical scarlet fever are more readily explainable on the basis of lack of sensitivity than by reason of an inherited immunity, since many of the mothers themselves are not immune. Recently, the great prevalence of hemolytic streptococci in the common infections of the upper respiratory tract of infants, such as

2. A résumé of these views will be found in the monograph on "Scarlet Fever" by A. Schlossmann and S. Meyer in Pfaundler and Schlossmann: Handbuch der Kinderheilkunde, ed. 3, Leipzig, F. C. W. Vogel, 1923, vol. 2, p. 101, and in a paper by Bristol, L. D.: Scarlet Fever as a Reaction of Hypersensitiveness to Streptococcus Protein, Am. J. M. Sc. **166**:853 (Dec.) 1923.

rhinitis, pharyngitis and otitis, is being recognized more and more as bacteriologic studies are made. The opportunity to acquire such an infection during infancy and a consequent specific protein hypersensitivity, appears almost universal, and the gradual development of skin sensitivity in later infancy accords with this view. In the development of sensitivity in the infant's skin, it is noticeable that the sensitivity to larger amounts of the filtrate appears earlier than the reaction to smaller amounts. This could readily be interpreted as indicating an increasing sensitivity of the skin cells to the antigen.

If one can assume that the skin reactions to scarlatinal streptococcus filtrate depend on a specific hypersensitivity, the possibility of a natural or hereditary allergic state, similar to the allergy to certain animal and plant protein antigens, might be considered. The lack of sensitivity in early life does not exclude such a contingency, since allergic persons usually do not exhibit hypersensitivity until after infancy. The widespread distribution of the sensitiveness to streptococcus filtrate, however, as well as the ease with which subjects can be immunized, is at variance with observations on other human hereditary allergic hypersensitivity. An acquired anaphylaxis to streptococcus protein would seem more in harmony with the present conception of hypersensitiveness than a natural allergy.

Probably the most striking analogy that favors the toxin theory is the demonstration of a neutralizing substance in the blood in many persons whose skin is relatively immune to the action of the filtrate. The relation of circulating, neutralizing antibody (antitoxin) to the skin reaction in older children and adults is apparently almost constant. In practically all those who do not react to small amounts of filtrate, this neutralizing substance is demonstrable, and the similarity of this to the inhibition of the Schick test by the presence of diphtheria antitoxin in the blood is apparent. In spite of this analogy, the explanation of this phenomenon as a toxin-antitoxin reaction is not the only possible one, since other antigen-antibody reactions are well known. One of these which has also many points of similarity to the human reactions with scarlatinal streptococcus filtrate is the protection which circulating homologous antibody affords sensitized guinea-pigs against anaphylactic shock. While no human counterpart of this particular antigen-antibody reaction is known, a brief discussion of it may serve to point out the possible application of this principle to the phenomenon associated with the streptococcus filtrate skin reactions and scarlet fever.

It has been shown by Weil [3] that in sensitized guinea-pigs the presence of an excess of circulating homologous antibody prevents the

3. Weil, R.: The Nature of Anaphylaxis and the Relations Between Anaphylaxis and Immunity, J. M. Research, New Series **22**:497 (March) 1913.

development of anaphylactic shock. This protection is apparently due to the union of the circulating antibody with the antigen before the latter reaches the susceptible cells. In Weil's experiments rather large amounts of antibody were necessary for the protection, since it required 3.5 cc. of immune serum to protect sensitive guinea-pigs against from 10 to 20 lethal doses of antigen. He explained this by an increased avidity of the sensitized cells for antigen as compared with the circulating antibodies, although Zinsser [4] believed that the antigen and antibody in the circulation are prevented from uniting quickly by some colloidal protective action of other serum constituents. The principle of protection of sensitive cells by homologous circulating antibody, therefore, is established, although the antigen seems to combine more quickly with the sessile cellular antibodies than with the free antibodies unless the latter are present in excess. It was further shown by Weil that this "masking" of the sensitivity by circulating homologous antibody in immune animals disappears, and the hypersensitivity returns when the excess of antibody is removed from the circulation. He concluded that "exactly the same antibodies are present in anaphylaxis as in immunity. In the former they predominate in the cells—in the latter, in the serum."

This principle of "masked anaphylaxis" may be tentatively applied to the relative skin immunity to streptococcus filtrate which occurs in the presence of circulating homologous neutralizing antibody in older children and adults. In such persons, variations in the quantity of filtrate antigen necessary to produce a positive skin reaction might easily depend on the degree of masking of the skin sensitivity by humoral antibodies. Those who have more circulating antibody would be expected to require more antigen to cause a positive reaction because of the necessity of neutralizing the local antibodies by the antigen before it could come in contact with the sensitized skin cells. This is supported by the regularity with which neutralizing "antitoxin" could be shown in those who were more immune and who gave negative skin reactions to considerable amounts of the toxin; the fact that such antitoxin was not always present in an amount sufficient to be demonstrated in those with less immunity whose skin was sensitive to relatively small amounts of the filtrate; and the observation that the introduction of serum containing antitoxin is followed by a decreaesd sensitivity of the skin in the recipient. Indeed, the protection of sensitive cells by the humoral antibody in this human hypersensitivity is apparently more in harmony with the theoretical considerations involved than the experimental results in guinea-pigs.

In comparing certain human anaphylactic manifestations with those of experimental animals such as the guinea-pig, the development of

4. Zinsser, H.: Infection and Resistance, ed. 3, New York, The Macmillan Company, 1923, p. 453.

symptoms seems to follow a relatively longer interval after the introduction of the antigen in man. If one disregards the forms of allergy apparently hereditary, and considers the type of acquired hypersensitivity which follows the injection of horse serum, for example, this interval is usually several days. Serum sickness is generally accepted as a human anaphylactic response, although of a type not identical with that in animals. In man, the anaphylactic reaction of serum sickness persists for a longer time, usually several days while in animals not fatally poisoned, the symptoms disappear relatively quickly. A slow and continuous combination of antigen with the cellular antibodies may be more characteristic of anaphylaxis in man. If this is true, the delay in reaction between the antigen and the body cells should offer a more favorable opportunity for the circulating antibodies to fix the antigen. In connection with the differences between human and animal anaphylaxis, a

Diagrammatic Scheme of Possible "Masked Anaphylaxis" to Scarlatinal Streptococcus Filtrate Protein

	Circulating Antibody Absent			Circulating Antibody Present (Masked Sensitivity)	
Skin Tests	No Sensitivity	Slight Sensitivity	Marked Sensitivity	Moderate Immunity	Marked Immunity
Negative to large amounts of antigen (50 STD)	Group 1	.....	.....	.....	Group 5
Negative to moderate amounts of antigen (20 STD)	.....	Group 2	.....	Group 4	.....
Positive to small amounts of antigen (2 STD or less)	.....	.....	Group 3	.....	.....

statement by Zinsser [5] regarding the species relationship between cellular and circulating antibodies might be quoted, viz.:

> Man seems to be an animal in which sensitiveness to the anaphylactic experiment is fortunately considerably less than in other animals, for reasons perhaps associated with peculiarities of antibody formation, or with the relative distribution of antibodies between cells and circulation.

The suggested application of the hypothesis of hypersensitivity and masked anaphylaxis to streptococcus protein, based on the skin reactions and antibody determinations reported in this study, might be illustrated by such a diagram as that indicated in the table. In this, only the differences in skin sensitivity to certain arbitrarily chosen amounts of filtrate have been illustrated as representative, although, obviously, intermediate degrees of sensitivity occur. The subjects in group 1 who gave negative reactions to rather large amounts of antigen and who were without circulating antibody might be considered to be unsensitized. Most young

5. Zinsser, H. (footnote 4, p. 476).

infants are in this group, although in some at this time of life there is apparently circulating antibody derived from an immune mother. Those in group 2, who were without humoral antibody and whose skin was sensitive to a moderate amount of filtrate, may represent a developing sensitivity. Such individuals are found in later infancy. The subjects in group 3, who had fully developed, well marked skin sensitivity and who were without circulating antibody, and those in groups 4 and 5 with immunity or a sensitivity masked by demonstrable circulating antibody and found during later childhood, have been discussed previously. Groups 1 and 2, therefore, would represent unsensitized or only partially sensitized persons without antibody; group 3, those in whom the antibody predominates in the cells, and groups 4 and 5, the persons in whom the antibody predominates in the serum.

It is possible that in older children and in adults the circulating antibody may vary in amount at different times, possibly increasing as a result of absorption of antigen from streptococcus infections or decreasing when such infections do not occur. The disappearance of the protecting antibody under the latter condition may unmask the hypersensitivity and account for the not infrequently observed development of scarlet fever in persons who had been previously clinically immune. It is also possible to explain how persons who are hypersensitive may not constantly develop clinical scarlet fever even when infected with the specific organisms. If the antigen is absorbed relatively slowly, a desensitization may occur without general toxic symptoms or rash. It has been demonstrated that in persons whose skin is sensitive, the injection of the scarlatinal streptococcus filtrate, at first in small amounts and then in gradually increasing doses, is followed by a rather rapid loss in sensitivity of the skin.

The applications of the hypothesis outlined to the effect of convalescent scarlet fever serum and scarlatinal antitoxin from immunized horses, on clinical scarlet fever, are obvious. Both the local and general action as illustrated by the Schultz-Charlton extinction test and therapeutic administration of serum are effective only when given relatively early, presumably before the reaction of the cells to the antigen is complete and has reached its height. The effect is that of inhibiting further reaction and the relatively rapid (twelve to twenty-four hour) disappearance of the evidences of specific toxicity. If, however, the serum is not given until the cellular reaction has become maximum, as evidenced, for example, by the rash having passed its fastigium, an effect is not demonstrable. It has been mentioned previously that such an anaphylactic manifestation as serum sickness, while abrupt in onset, tends to pursue a somewhat prolonged course, so that the cellular injury appears to be gradual and continuous rather than immediately maximum.

In this connection, the observations first made by Trask [6] and Blake and Trask [7] and confirmed by my own, are especially striking. In studies on the presence of toxin (antigen) demonstrable in the circulation during clinical scarlet fever, it was found that the amount of toxin had practically no relation to the clinical severity of the toxemia, and surprisingly large amounts could at times be demonstrated in the serum in clinically mild cases. Also, during the second week, when the rash had faded, circulating toxin was found, indicating that the disappearance of the rash is not dependent on the formation of antitoxin. Such observations are in harmony with variations in hypersensitivity, and the presence of circulating antigen after the saturation of cellular antibodies.

Attention might also be called to a local "toxin-extinction" of the rash in scarlet fever noted by Zoeller,[8] who describes the effect of the intradermal injection of Dick toxin in early scarlet fever rashes. This results in a primary marked local accentuation of the skin rash which lasts for forty-eight hours and is followed on the third day by local blanching of the injected skin area. In older rashes, the changes described did not occur. Such an accelerated local exhaustion of the cellular antibodies by an excess of antigen would be expected in sensitized cells.

The question of the specificity of certain strains of hemolytic streptococci in causing scarlet fever is one about which conclusive proof seems to be lacking. It is true that a majority of those strains isolated from patients with scarlet fever have been found to have similar biologic characteristics, such as specific agglutinability, as compared with strains from nonscarlatinal sources (Bliss, Gordon, Tunnicliff, Rosenow, Stevens and Dochez and others).[9] On the other hand, strains from scarlet fever vary in their ability to ferment certain sugars (the Dicks)[10]; and the production of the presumably characteristic and specific toxin which causes skin reactions in persons with a positive reaction to the Dick test and which is neutralizable by immune scarlatinal serum, is apparently a function of a large number of strains isolated from non-

6. Trask, J. F.: Studies in Scarlet Fever: 1. The Amount of Scarlet Fever Toxin in the Blood of Patients with Scarlet Fever, J. Clin. Investigation **3**:391 (Dec.) 1926.

7. Blake, F. G., and Trask, J. D.: Studies in Scarlet Fever: II. The Relation of the Specific Toxemia of Scarlet Fever to the Course of the Disease, J. Clin. Investigation **3**:397 (Dec.) 1926.

8. Zoeller, C.: La toxi-extinction de l'exanthème scarlatineux, Comp. rend. Soc. de biol. **92**:337 (Feb. 13) 1925.

9. Tunnicliff, R.: The Identification of the Streptococcus of Scarlet Fever, J. A. M. A. **87**:625 (Aug. 28) 1926.

10. Dick, G. F., and Dick, G. H.: The Etiology of Scarlet Fever, J. A. M. A **82**:301 (Jan. 26) 1924.

scarlatinal sources (Parhaf,[11] Rosenow,[12] and Kirkbride and Wheeler [13]). The same variation in ability to produce toxin in vitro occurs in strains isolated from patients with clinical scarlet fever as in those from other sources. In some a skin reaction is given by 0.00001 cc. of the filtrate, while with other strains 0.01 cc. is required. It has been suggested that many such infections with toxin-producing organisms occur in persons with an immunity to the scarlatinal rash and are in reality *scarlatina sine exanthemate*. This seems probable, although if all such strains are true scarlatinal streptococci, there is an unexpectedly large and widespread distribution of such specific scarlet fever organisms. Variability in the formation of antigen by various strains as well as variations in immunity of persons may play a part in the toxic clinical manifestations of scarlet fever.

The increasing number of observations on the occurrence of infections with toxin-producing hemolytic streptococci in persons without a rash, previously mentioned, indicates that there are two distinct phases in infection with such organisms. First, contact infection with invasion of the tissues, usually faucial and pharyngeal, occurs and is dependent on conditions of individual resistance at present unknown. After this infection, the specific toxic syndrome (scarlet fever) from absorption of the streptococcus protein (toxin) appears only in those persons who may be anaphylactically hypersensitive to this protein. Others, in whom this hypersensitivity may be masked by specific circulating antibody, are immune to the specific intoxication and present only the nonspecific phenomena of pyogenic infection. The fact that such infections occur in persons with readily demonstrable neutralizing antibody in the blood makes it unlikely that this substance protects from infection itself. The protection which is afforded by the specific antitoxin, therefore, seems to be limited to the specific toxic phenomena, and the toxic factor in scarlet fever, which may be anaphylactic, must be distinguished from the septic factor.

In the foregoing discussion are detailed certain considerations on which is based presumptive evidence of an important factor of hypersensitiveness in scarlet fever. The increasing difficulties in applying the soluble toxin theory to the phenomena observed in connection with the disease warrant the serious consideration of another hypothesis.

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11. Parhaf, J.: Recherches sur la toxine streptococcique et la réaction de Dick, Bull. et mém. Soc. méd. d. hôp. de Paris **49**:395 (March 13) 1925.

12. Rosenow, E. C.: The Precipitin Reaction in the Diagnosis of Scarlet Fever and Allied Hemolytic Streptococcus Infections, J. A. M. A. **83**:9 (Jan. 2) 1926.

13. Kirkbride, M. B., and Wheeler, M. W.: Further Observations on the Toxins of Hemolytic Streptococci, J. Immunol. **13**:19 (Jan.) 1927.

THE HEARTS OF NORMAL CHILDREN

III. ELECTROCARDIOGRAPHIC RECORDS *

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The cardiograms which represent the basis of this study are records of the same school children whose clinical observations and teleoroentgenograms were reported in previous articles. Many of the cardiograms are in series; in a group of sixty-three children, from four to seven tracings were made in as many successive years, so that we had an excellent opportunity to study serial characteristics. As our group consisted of 222 normal school children from 3 to 12 years old, and was many times larger than any which had been used in previous studies, we endeavored to establish a series of normal figures for each age period, with standard deviations, so that the limits of the normal might be appreciated. In addition, we tried to show possible sex and family characteristics in the cardiograms, and to stress the differences between the tracings of an adult's heart and those of a child's heart.

All the cardiograms were made under usual conditions, with the child in a sitting position. The children were taken to the office in small groups, usually of friends, and every effort was made to remove any element of fear. All measurements have been taken from the base line of the string. All the deflections were measured, as well as the intervals, and tabulated according to age and sex. In addition, the pulse rate and incidence of sinus arrhythmia were studied and tabulated. We were especially interested in the rate of change of the deflections as found in the long series of electrocardiograms from the same children, and in the marked similarities in the tracings found in some siblings. The electrocardiograms showing so-called ventricular preponderance were also of interest, especially when related to clinical and roentgen-ray observations. It is interesting to note that abnormal rhythms, premature contractions and inverted T waves were not found in the first or second leads in these examinations.

In 1908, Nicolai and Funaro [1] showed the frequency of the deep S wave in the first lead in infants. They studied forty-five chil-

* Received for publication, Dec. 19, 1927.

* From the Bureau of Educational Experiments.

1. Nicolai and Funaro: Das Elektrokardiogram des Säuglings, Zentralbl. f. Physiol. **22**:58, 1908. Funaro, R.: L'Ellettro-cardiogramma dell'Adulto e del Bambino, Riv. di clin. pediat. **8**:480 (June) 1910.

dren mostly infants but fail to state whether all their cases were normal, and they used only the first lead. Later in the same year, the deep S wave was noted by Heubner [2] who, in addition, drew attention to the small or absent R wave in the first lead in infants. He also took tracings only of the first lead. In 1913, Hecht [3] made a comprehensive study of the electrocardiogram in the child, and came to the conclusion that all the deflections were smaller in childhood and that the transmission time was shorter than in adults. In 1916, Krumbhaar and Jenks,[4] in a study of forty-five children, showed a preponderance of the right ventricle in young infants and a decrease in transmission time in childhood. They also found that the average ventricular deflections were greater in children than in adults and that sinus arrhythmia became increasingly more frequent from the sixth year to the age of puberty.

In 1921, Seham [5] made an intensive study of the electrocardiogram in infancy and childhood. He was the first to establish a composite curve as a guide for the normal at all ages of childhood, and confirmed the observations of Krumbhaar and Jenks regarding the greater size of deflections and shorter transmission time in children. Unfortunately, he did not make differentiations as to sex in stating his results. He studied new-born children and infants under 1 year of age, and, in addition, thirty-six children from 2 to 13 years old.

SINUS ARRHYTHMIA

Arrhythmia becomes more pronounced as the child grows older and the occurrence of sinus arrhythmia varies inversely with the rapidity of the pulse rate. Seham also holds this view, but previous workers found that arrhythmias were more common in the infant than in later childhood.

Wiggers [6] suggested that this variation is produced when the vagi periodically inhibit the rate of impulse formation in the sinus node, but, on the other hand, Bass [7] expressed the opinion that the arrhythmia

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2. Heubner, O.: Das Elektrokardiogram des Säuglings and Kindes, Monatschr. f. Kinderh. **7**:6 (April) 1908.

3. Hecht, A. F.: Der Mechanismus des Herzaktion im Kindesalter, Ergebn. d. Inn. Med. u. Kinderh. **11**:324, 1913.

4. Krumbhaar, E. B., and Jenks, H.: Electrocardiographical Studies in Normal Infants and Children, Heart **6**:189, 1916.

5. Seham, M.: Electrocardiography in Children, in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1924, vol. 4, p. 282.

6. Wiggers, C. J.: Physiology of Circulation, in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1924, vol. 4, p. 198.

7. Bass, M. H.: Acquired Diseases of the Heart, in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1924, vol. 4, p. 344.

is due to variation in the length of the diastole. Krumbhaar [4] designated as a sinus arrhythmia a rhythm in which the difference between the longest and shortest pulse period interval was 0.1 second or more. Using this rule as our standard, we found that an average of 62 per cent of our patients showed a sinus arrhythmia with an average pulse period difference of 0.145 second. This statement brings out the comparative lack of value of statistics gathered as averages only, without differentiation

TABLE 1.—*Percentage of Children Showing Arrythmia at Various Ages*

Age	Boys	Girls
3	44	..
4	72	54
5	50	58
6	66	52
7	45	54
8	71	70
9	64	81
10	63	72
11	68	86
12	87	78

TABLE 2.—*Arrhythmia, Showing Average Pulse Period Differences at Various Ages*

	Boys			
Age	Number of Cases	Average	± Standard Deviation	Range
3	9	0.11	0.10	0.02–0.30
4	18	0.12	0.07	0.00–0.26
5	24	0.11	0.07	0.00–0.26
6	29	0.13	0.06	0.04–0.27
7	33	0.12	0.10	0.00–0.56
8	31	0.16	0.10	0.04–0.32
9	33	0.13	0.07	0.02–0.40
10	27	0.15	0.08	0.04–0.34
11	19	0.16	0.10	0.04–0.40
12	15	0.17	0.10	0.04–0.44
	Girls			
Age	Number of Cases	Average	± Standard Deviation	Range
3	..	....	....	.....
4	22	0.12	0.07	0.00–0.28
5	33	0.13	0.12	0.00–0.44
6	29	0.13	0.10	0.04–0.48
7	35	0.15	0.11	0.00–0.40
8	30	0.16	0.10	0.02–0.44
9	26	0.16	0.09	0.04–0.38
10	18	0.16	0.07	0.04–0.28
11	21	0.19	0.11	0.08–0.56
12	9	0.18	0.13	0.04–0.48

as to sex or age, for on further analysis we found a pulse period difference of 0.136 second for boys and one of 0.153 second for girls, giving an entirely different picture of arrhythmia in boys than in girls. Again, when we studied the individual age groups we found that an average arrhythmia stated for all ages from 2 to 12 years had little clinical significance, as is shown in table 1. It is seen at once that arrhythmia occurs less frequently in early childhood, although the increase is irregular. The greatest arrhythmia is evident from the eighth to the twelfth years of age. As is seen in table 2, the pulse period difference increases steadily with age.

The longest arrhythmic period in our series was 0.56 second, found in a boy, aged 7, and in a girl, aged 11. It is interesting to note that both these children had the slowest pulse rates in their age groups: the boy, a pulse rate of 74, the average for his age group being 90, and the girl a pulse rate of 65, the average for her age group being 87. Their electrocardiograms did not show any other resemblances except for the arrhythmia.

The electrocardiographic arrhythmias are considerably fewer in number than those noted in the clinical observations. Perhaps the tense

TABLE 3.—*Average Measurements of Heart Rate with Standard Deviations*

Age	Number of Cases	Average Rate Electrocardiogram	± Standard Deviation	Range	Vertical Heart Rate (Auscultation)
Boys					
3	9	113.1	10.7	100–134	102
4	18	104.3	12.1	85–135	100
5	24	106.0	13.4	84–135	95
6	29	100.2	13.2	75–126	95
7	33	100.1	14.0	74–122	92
8	31	96.2	14.7	75–135	90
9	33	96.4	11.8	72–120	88
10	27	92.3	11.3	70–109	89
11	19	89.4	9.2	75–104	85
12	15	81.7	14.9	65–100	82
Girls					
3	..	.....	....	.....	..
4	22	113.4	12.8	88–144	99
5	33	107.9	10.2	80–124	99
6	29	103.1	9.2	81–124	94
7	35	98.3	11.7	73–126	92
8	30	90.0	11.2	71–110	90
9	26	90.0	9.7	75–110	89
10	18	91.6	18.0	71–133	91
11	21	87.0	10.9	65–110	89
12	9	86.3	12.3	74–105	82

or excited child in the environment of the electrocardiographic room has a more rapid pulse and less arrhythmia than on ordinary physical examination, or perhaps the clinician can note as an arrhythmia a pause of less than 0.1 second. When all arrhythmia measurable on the cardiogram was taken into account, including pulse period differences of less than 0.1 second, only ten children showed absolutely regular pulse rates, and these were in the younger groups, aged 7 years or under, with pulse rates of 100 or more a minute.

HEART RATE

The heart rate becomes slower as the child grows older. The decrease in rate is not as regular as that of the vertical pulse rate taken at the time of the physical examination, and the heart rates taken from the cardiograms are slightly more rapid, a difference probably due to

some fear of the procedure of making a cardiogram. This supposition is strengthened by the fact that the younger groups showed a greater difference in rate between clinical and electrocardiographic observations than the older groups, as shown in table 3.

Girls have a greater heart rate than boys up to the age of 7. After this age, boys have more rapid heart rates than girls until the age of 12; then girls again have the higher rate. No child under the age of 6 years had a rate slower than 80. The slowest rate found in the series was a rate of 58 in a girl, aged 10, whose examinations and cardiograms were not otherwise noteworthy. Her cardiogram showed an arrhythmia with an average pulse period difference of 0.2 second. The most rapid

TABLE 4.—*Transmission Time of Normal Children P-R Interval*

Boys				
Age	Number of Cases	Average	± Standard Deviation	Range
3	9	0.13	0.04	0.12-0.15
4	18	0.13	0.03	0.10-0.16
5	24	0.14	0.02	0.12-0.17
6	29	0.13	0.02	0.11-0.17
7	33	0.14	0.02	0.11-0.19
8	31	0.14	0.02	0.10-0.18
9	33	0.13	0.01	0.10-0.16
10	27	0.14	0.01	0.10-0.18
11	19	0.14	0.01	0.12-0.16
12	15	0.14	0.04	0.12-0.18

Girls				
Age	Number of Cases	Average	± Standard Deviation	Range
3	..	....	....	.....
4	22	0.13	0.01	0.10-0.16
5	33	0.12	0.02	0.10-0.16
6	29	0.13	0.01	0.12-0.16
7	35	0.14	0.02	0.11-0.16
8	30	0.14	0.01	0.12-0.16
9	26	0.14	0.02	0.12-0.16
10	18	0.14	0.02	0.12-0.16
11	21	0.14	0.02	0.12-0.16
12	9	0.14	0.02	0.12-0.16

rate, 144, was found in a girl, aged 4, who continued to have a rapid rate, 124, the following year; at the age of 6 years, however, her rate was the average, and it has continued to be normal since that time. She has never shown any other abnormal signs. She had a slight sinus arrhythmia with an average pulse period difference of only 0.04 at the time her heart rate was 144; two years later she had a normal heart rate and an arrhythmia of 0.08.

P-R INTERVAL

The period of time taken for the stimulus to pass from the sino-auricular node through the auricles and through the auriculoventricular node is represented by the P-R interval. Lewis and Gilder [8] found the

8. Lewis, T., and Gilder, M. D.: The Human Electrocardiogram and Preliminary Investigations of Young Male Adults to Form Basis for Pathological Study, Phil. Tr. Roy. Soc. London series B **202**:351, 1912.

normal limits in young adults to be 0.13 second to 0.21 second with an average of 0.18 second. We found a more rapid conduction time for children with an average of 0.136 second, our figures agreeing closely with those of Seham and Hecht. Hecht suggested that the P-R interval is shorter in childhood because the differentiation of the auriculoventricular bundle is not complete before adolescence.

Q-R-S INTERVAL

The Q-R-S interval represents the period of time in which the stimulus passes into the ventricles from the auriculoventricular node

TABLE 5.—*Transmission Time of Normal Childrens Q-R-S Interval*

		Boys		
Age	Number of Cases	Average	± Standard Deviation	Range
3	9	0.06	0.01	0.04–0.08
4	18	0.06	0.02	0.04–0.08
5	24	0.06	0.01	0.04–0.09
6	29	0.06	0.01	0.04–0.08
7	33	0.06	0.01	0.04–0.08
8	31	0.06	0.01	0.04–0.08
9	33	0.06	0.01	0.04–0.08
10	27	0.07	0.01	0.04–0.08
11	19	0.06	0.01	0.04–0.08
12	15	0.07	0.01	0.06–0.08
		Girls		
Age	Number of Cases	Average	± Standard Deviation	Range
3	..	....	....	.....
4	22	0.06	0.08	0.04–0.08
5	33	0.06	0.02	0.04–0.08
6	29	0.06	0.02	0.04–0.10
7	35	0.06	0.01	0.04–0.08
8	30	0.06	0.01	0.04–0.08
9	26	0.06	0.01	0.04–0.08
10	18	0.06	0.01	0.05–0.08
11	21	0.06	0.01	0.04–0.08
12	9	0.06	0.01	0.04–0.08

through the bundle of His and its branches. Lewis [9] termed this period "The stage of invasion" and gave 0.10 second as the upper normal limit of time for the event in young adults. Again we find a shorter conduction time in children, approximately 0.06 second for the ages studied, with no significant age differences, as shown in table 5. Seham's average for children in his series from 2 to 3 years of age exactly accords with this.

S-T TRANSITION

In children, the T wave slopes most frequently directly from the downstroke of the R wave or upstroke of the S deflection, making it impossible, usually, to find any dividing interval. Lewis [9] found this iso-electric period, which he termed the "stage of possession," generally

9. Lewis, Thomas: Graphic Registration of the Heart Beat, ed. 3, London, Shaw & Sons, 1925.

short, and often absent, in the adult. Seham gave an average of 0.213 seconds for the duration of the S-T interval in children from 1 to 5 years of age, and 0.273 seconds in those from 6 to 13 years. Oppenheimer said that there is no measureable S-T transition, and we also were frequently unable to measure it on the cardiograms in our series.

P DEFLECTION

The chief deflection of the auricular complex is represented by the P wave in the electrocardiogram. No significant changes with increasing

TABLE 6.—*The P Deflection*

Boys										
		P_1			P_2			P_3		
Age	Number of Cases	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range
3	9	0.15	0.06	0.05–0.20	0.21	0.09	0.10–0.45	0.12	0.10	0.00–0.35
4	18	0.13	0.05	0.05–0.20	0.17	0.03	0.10–0.30	0.09	0.05	0.00–0.20
5	24	0.12	0.02	0.00–0.30	0.18	0.08	0.00–0.35	0.12	0.07	0.00–0.25
6	29	0.14	0.07	0.05–0.30	0.17	0.07	0.10–0.35	0.10	0.07	0.00–0.25
7	33	0.12	0.05	0.05–0.30	0.15	0.06	0.05–0.30	0.09	0.07	0.00–0.30
8	31	0.11	0.07	0.00–0.25	0.16	0.08	0.00–0.30	0.11	0.08	0.00–0.25
9	33	0.12	0.05	0.00–0.20	0.16	0.06	0.05–0.25	0.10	0.05	0.00–0.20
10	27	0.15	0.06	0.10–0.25	0.17	0.06	0.05–0.30	0.10	0.06	0.00–0.25
11	19	0.13	0.05	0.05–0.25	0.17	0.06	0.10–0.30	0.11	0.07	0.00–0.20
12	15	0.16	0.07	0.10–0.25	0.17	0.04	0.05–0.30	0.13	0.08	0.00–0.25

Girls										
		P_1			P_2			P_3		
Age	Number of Cases	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range
3	..	....	....	.....	....	....	.....	....	....	.....
4	22	0.15	0.03	0.05–0.30	0.17	0.07	0.10–0.30	0.09	0.07	0.00–0.30
5	33	0.13	0.07	0.05–0.30	0.17	0.08	0.00–0.30	0.11	0.09	0.00–0.30
6	29	0.10	0.02	0.05–0.25	0.14	0.05	0.05–0.25	0.08	0.06	0.00–0.20
7	35	0.12	0.05	0.05–0.25	0.17	0.05	0.10–0.30	0.12	0.06	0.00–0.30
8	30	0.15	0.06	0.05–0.25	0.18	0.07	0.00–0.30	0.12	0.05	0.00–0.25
9	26	0.13	0.06	0.00–0.20	0.18	0.08	0.00–0.30	0.12	0.07	0.00–0.25
10	18	0.15	0.05	0.05–0.25	0.18	0.06	0.10–0.30	0.14	0.09	0.00–0.55
11	21	0.15	0.07	0.05–0.30	0.17	0.06	0.10–0.30	0.11	0.05	0.00–0.20
12	9	0.13	0.02	0.10–0.20	0.14	0.07	0.05–0.25	0.11	0.08	0.00–0.29

age in the height of this deflection were found in our groups, although the wave throughout tended to be higher than in the adult. This was also noted by Seham, and suggests that the summation of action currents in the child's auricle is relatively greater than it is in the adults. The P deflection in leads I and II we found to be uniformly larger than in lead III. Lewis and Gilder stated that this is true for adults, but Seham found that the P wave was smallest in lead I.

ABERRANT FORMS OF P WAVES

Although cases have been reported in which the P deflection in the first or second lead was found inverted, abnormal P waves were not noted in the first lead in our series, and there was only one instance in

which a P_2 was inverted. This was in a boy, aged 12 years, whose P waves in the second lead had previously been small and whose P waves in the third lead showed aberrant forms in four successive years. A negative P_2 was found in our records only in rare instances. P_3 was inverted or diphasic in fifty-two, or 10 per cent, of our electrocardiograms; of these 3.2 per cent showed a constant inversion of the P in lead III; 1.8 per cent showed a constant diphasic P_3, and 2.8 per cent showed an occasional diphasic P_3. Goddard [10] has reported finding P_3 inverted in about 10.8 per cent of his cases. In Seham's group of thirty-six children from 2 to 13 years old, he found only two diphasic or inverted P waves.

Lewis [9] has shown that changes in the P wave in human curves are largely controlled by the vagus. He explained conspicuous changes in the P wave during expiration as being due to vagal inhibition displacing the pacemaker. Some change is also induced occasionally by the rotation of the heart on its own axis, while the inconstant irregularities are possibly respiratory in origin.

It is interesting to note that in fourteen of our cases the inversion was found in serial years; and in four families the condition was present in two or more members. Twenty-seven cases, or 55 per cent, of the series showing aberrant P_3's, showed a splitting or notching of the R_3 or S_3 following the inverted P_3. In these cases, the commencing limb of the inverted deflection usually appeared later than that of the upright P's in the other leads, so that the P-R interval was shortened. It is noteworthy that none of the children who showed aberrant auricular deflections had large hearts as shown by the teleoroentgenogram.

Q-R-S COMPLEX

The Q-R-S deflections correspond to the composite superimposed activities of both ventricles during the invasion of the excitation wave of systole. In our series, we frequently found split or deeply notched Q-R-S curves. This possibly indicates that in the child the small units of which the ventricles are composed are less unified than in the adult.

THE Q DEFLECTION

The beginning of ventricular impulse conduction is represented by the Q wave. It has been shown that Q in the first lead is due to left ventricular activity and is a right ventricular event in leads II and III.

The Q wave was not constant in our cases. When present it was usually of a much greater degree than in adults, suggesting a greater intensity of early ventricular activity. The Q deflection gave a greater average depth in boys than in girls and Q waves decreased in height with

10. Goddard, quoted by Seham (footnote 3, p. 289).

age. Q_3 was split in the tracings of two children with no symptoms nor signs suggesting cardiac disease. Q_1 and Q_2 were never found notched or split.

Krumbhaar found the Q wave relatively deep in childhood. Seham found that the depth of Q_2 and Q_3 was in general greater than in adults; he found Q_1 absent in premature infants and variable and indefinite until the fifth month, then gradually increasing until the fifth year, when it appeared as in adults. This, he believed, corroborated Lewis' theory that Q_1 represented left ventricular activity.

TABLE 7.—*The Q Deflection*

Boys

Age	Number of Cases	Q_1 Average	Q_1 ± Standard Deviation	Q_1 Range	Q_2 Average	Q_2 ± Standard Deviation	Q_2 Range	Q_3 Average	Q_3 ± Standard Deviation	Q_3 Range
3	9	0.13	0.12	0.00–0.30	0.13	0.11	0.00–0.30	0.22	0.18	0.00–0.60
4	18	0.10	0.10	0.00–0.30	0.08	0.11	0.00–0.40	0.10	0.17	0.00–0.70
5	24	0.08	0.09	0.00–0.30	0.11	0.11	0.00–0.40	0.15	0.15	0.00–0.55
6	29	0.09	0.07	0.00–0.20	0.09	0.10	0.00–0.40	0.15	0.12	0.00–0.50
7	33	0.08	0.09	0.00–0.35	0.08	0.10	0.00–0.30	0.10	0.13	0.00–0.40
8	31	0.08	0.09	0.00–0.30	0.08	0.09	0.00–0.25	0.10	0.13	0.00–0.50
9	33	0.07	0.08	0.00–0.40	0.07	0.09	0.00–0.35	0.11	0.14	0.00–0.60
10	27	0.11	0.12	0.00–0.60	0.07	0.07	0.00–0.25	0.11	0.13	0.00–0.50
11	19	0.07	0.08	0.00–0.30	0.05	0.06	0.00–0.25	0.08	0.09	0.00–0.30
12	15	0.08	0.08	0.00–0.30	0.04	0.05	0.00–0.15	0.05	0.07	0.00–0.20

Girls

Age	Number of Cases	Q_1 Average	Q_1 ± Standard Deviation	Q_1 Range	Q_2 Average	Q_2 ± Standard Deviation	Q_2 Range	Q_3 Average	Q_3 ± Standard Deviation	Q_3 Range
3	..	....	....	.....	....	....	.....	....	....	.....
4	22	0.07	0.08	0.00–0.35	0.08	0.08	0.00–0.20	0.15	0.13	0.00–0.40
5	33	0.09	0.06	0.00–0.40	0.07	0.08	0.00–0.35	0.12	0.15	0.00–0.50
6	29	0.07	0.08	0.00–0.30	0.05	0.07	0.00–0.30	0.10	0.08	0.00–0.40
7	35	0.05	0.07	0.00–0.25	0.04	0.07	0.00–0.30	0.07	0.10	0.00–0.30
8	30	0.07	0.11	0.00–0.50	0.05	0.06	0.00–0.20	0.05	0.07	0.00–0.30
9	26	0.06	0.07	0.00–0.20	0.05	0.07	0.00–0.20	0.04	0.06	0.00–0.40
10	18	0.06	0.08	0.00–0.25	0.03	0.04	0.00–0.10	0.04	0.02	0.00–0.20
11	21	0.04	0.05	0.00–0.20	0.04	0.05	0.00–0.15	0.05	0.07	0.00–0.30
12	9	0.03	0.04	0.00–0.10	0.03	0.04	0.00–0.10	0.02	0.04	0.00–0.10

R DEFLECTION

It has been shown that the R wave of the ventricular complex corresponds to the activity of the main mass of the ventricular wall. The R wave is usually the greatest of the electrocardiographic deflections. We found it much higher in children than in adults, confirming Seham's observations in his group of children, aged from 2 to 13 years. This is the reverse of Linetzky's [11] observations that the R wave becomes

11. Linetzky, S.: Die Beziehungen der Form des Elektrocardiogramms zu dem Lebensalt, der Herzgrosse, und dem Blutdruck, Ztschr. f. exper. Path. u. Therap. **9**:669, 1911.

higher with age and with increase in the size of the heart. R_1 in our series is higher in boys than in girls aged from 6 through 12 years. In lead III, the R wave is less in boys than in girls until the age of 10. Lewis has shown that R is chiefly a left ventricular event in lead I and a right ventricular event in lead III. We found R_1 notched in only three cases, while R_2 was notched or split fifteen times and R_3 notched 113 and split 56 times among our 516 electrocardiograms. Lewis finds notched or bifurcated R_2's and R_3's infrequently in adults.

TABLE 8.—*The R Deflection*

		Boys								
		R_1			R_2			R_3		
Age	Number of Cases	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range
3	9	0.99	0.34	0.55–1.50	1.59	0.67	0.80–3.20	1.09	0.76	0.50–3.10
4	18	1.01	0.35	0.60–2.00	1.33	0.41	0.80–2.20	0.91	0.38	0.20–1.60
5	24	0.98	0.27	0.50–1.50	1.52	0.58	0.40–2.65	1.14	0.57	0.20–2.40
6	29	1.03	0.32	0.50–1.60	1.42	0.37	0.70–2.30	1.02	0.46	0.40–2.10
7	33	1.02	0.29	0.60–1.80	1.34	0.41	0.85–2.30	1.03	0.45	0.30–2.80
8	31	1.06	0.31	0.70–1.75	1.45	0.41	0.70–2.10	0.98	0.45	0.35–1.50
9	33	1.00	0.28	0.50–1.70	1.60	0.53	0.80–2.80	1.13	0.57	0.40–2.50
10	27	1.21	0.73	0.50–2.20	1.55	0.48	0.85–2.90	1.20	0.54	0.40–2.20
11	19	0.95	0.33	0.60–1.60	1.45	0.37	0.80–2.50	1.02	0.48	0.50–2.30
12	15	1.05	0.52	0.70–1.80	1.47	0.32	1.00–2.00	1.02	0.25	0.60–0.90
		Girls								
		R_1			R_2			R_3		
Age	Number of Cases	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range
3	..	....	....	.....	....	....	.....	....	....	.....
4	22	1.07	0.26	0.65–1.70	1.53	0.36	1.00–2.30	1.19	0.73	0.30–2.36
5	33	1.01	0.27	0.40–1.65	1.48	0.48	0.65–2.60	1.11	0.51	0.25–2.00
6	29	0.86	0.23	0.50–1.30	1.33	0.29	0.60–2.50	1.10	0.54	0.30–2.40
7	35	0.92	0.33	0.50–1.60	1.12	0.96	0.70–2.40	1.08	0.59	0.30–2.60
8	30	0.90	0.57	0.60–1.70	1.53	0.39	0.80–2.60	1.14	0.50	0.40–2.30
9	26	0.88	0.36	0.00–1.55	1.59	0.45	0.70–2.40	1.14	0.55	0.50–2.50
10	18	0.92	0.32	0.50–1.70	1.35	0.37	0.09–2.00	0.91	0.34	0.40–1.50
11	21	0.94	0.35	0.55–2.00	1.48	0.47	0.70–2.70	0.99	0.47	0.40–2.10
12	9	0.97	0.13	0.45–1.50	1.45	0.43	1.05–2.40	0.87	0.40	0.40–1.50

S DEFLECTION

The S deflection is not constantly found in the normal electrocardiogram. In lead I, S is a right ventricular event, in leads II and III it shows left ventricular activity. Lewis explained its presence as showing the spread of the excitation wave to the apex before reaching the base of the ventricles. He found it absent in basal priority. In our cases, the average S wave was less deep than in adults, but this was due to the fact that in many tracings the S wave was absent. When present, this deflection was deeper than it was in adults. The boys in our series gave deeper S waves than the girls. S_1 in boys and girls and S_2 in boys showed a decrease in size with increasing age, but S_3 in boys showed a gradual increase in depth with age, suggesting an increase in left ventricular activity. We found S_3 notched in twenty-two cases and split in six cases among our 516 electrocardiograms.

The presence of a deep S wave in infancy was established by Nicolai and Funaro and by Heubner, and Seham showed that the S wave reached its peak at the end of the third month, and then grew rapidly smaller until the sixth month, reaching its minimum deflection at the age of 2 years; then, after a short increase, it remained about the same throughout the rest of childhood. He found S_1 deeper than in the adult; S_2 and S_3 were generally lower in infancy than in childhood, the average of S_3 being lower than S_2.

TABLE 9.—*The S Deflection*

		Boys								
		S_1			S_2			S_3		
Age	Number of Cases	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range
3	9	0.29	0.20	0.00–0.65	0.30	0.20	0.10–0.70	0.17	0.21	0.00–0.70
4	18	0.22	0.18	0.00–0.60	0.29	0.12	0.10–0.50	0.25	0.29	0.00–1.10
5	24	0.18	0.10	0.00–0.45	0.24	0.18	0.00–0.70	0.11	0.12	0.00–0.40
6	29	0.22	0.18	0.00–0.60	0.20	0.15	0.00–0.55	0.14	0.18	0.00–0.90
7	33	0.15	0.14	0.00–0.50	0.18	0.13	0.00–0.49	0.16	0.17	0.00–0.75
8	31	0.16	0.13	0.00–0.50	0.21	0.17	0.00–0.45	0.16	0.15	0.00–0.55
9	33	0.23	0.17	0.00–0.60	0.23	0.16	0.00–0.55	0.15	0.16	0.00–0.60
10	27	0.17	0.20	0.00–0.95	0.18	0.14	0.00–0.55	0.19	0.21	0.00–0.90
11	19	0.16	0.12	0.00–0.40	0.18	0.10	0.00–0.40	0.18	0.18	0.00–0.50
12	15	0.17	0.13	0.00–0.40	0.20	0.16	0.00–0.60	0.20	0.19	0.00–0.90
		Girls								
		S_1			S_2			S_3		
Age	Number of Cases	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range	Average	± Standard Deviation	Range
3	..	....	....	.....	....	....	.....	....	....	.....
4	22	0.21	0.19	0.00–0.50	0.17	0.12	0.00–0.40	0.11	0.13	0.00–0.40
5	33	0.16	0.11	0.00–0.35	0.14	0.12	0.00–0.50	0.12	0.18	0.00–0.50
6	29	0.20	0.15	0.00–0.70	0.20	0.15	0.00–0.70	0.12	0.11	0.00–0.35
7	35	0.19	0.14	0.00–0.50	0.21	0.15	0.00–0.50	0.12	0.13	0.00–0.70
8	30	0.18	0.14	0.00–0.40	0.19	0.12	0.00–0.40	0.15	0.17	0.00–0.70
9	26	0.19	0.14	0.00–0.50	0.21	0.16	0.00–0.60	0.11	0.11	0.00–0.30
10	18	0.13	0.12	0.00–0.40	0.21	0.14	0.00–0.50	0.19	0.19	0.00–0.60
11	21	0.13	0.11	0.00–0.50	0.15	0.11	0.00–0.35	0.12	0.12	0.00–0.45
12	9	0.13	0.05	0.05–0.20	0.15	0.12	0.00–0.40	0.12	0.09	0.00–0.30

T WAVE

The usual final ventricular deflection is found constantly in children past the age of infancy. This wave has been shown to represent recovery from excitation, and is not a peculiar ventricular manifestation. The adult form is a gradual rise and steep fall. In the second lead, the T wave has been considered a gage of muscular activity. It is decreased in increased vagus action.[12] Our observations showed that the T wave was higher and broader in children than in adults, and higher and broader in boys than in girls. In the third lead in girls there

12. Rothberger and Winterberg: Arch. f. d. ges. Physiol. 1910, vol. 135, quoted in Sachs, H.: Ueber das Elektrokardiogramm der Schwangeren, Berl. Klinik. **57:** 803 (Aug. 23) 1920.

TABLE 10.—*The T Deflection*

Boys

Age	Number of Cases	T_1 Average	T_1 ± Standard Deviation	T_1 Range	T_2 Average	T_2 ± Standard Deviation	T_2 Range	T_3 Average	T_3 ± Standard Deviation	T_3 Range	Inverted T_3 Average	Inverted T_3 ± Standard Deviation	Inverted T_3 Range
3	9	0.36	0.09	0.20–0.50	0.34	0.17	0 10–0.70	0.21	0.04	0.10–0.40	0.11	0.04	0.05 0.15
4	18	0.34	0.12	0.10–0.60	0.37	0.11	0.10–0.55	0.14	0.06	0.00–0.25	0.11	0.04	0.05–0.20
5	24	0.31	0.14	0.00–0.65	0.39	0.16	0.05–0.70	0.20	0.13	0.00–0.40	0.13	0.08	0.05 0.25
6	29	0 37	0.13	0.20–0.60	0.39	0.08	0.20–0.55	0.21	0.12	0.00–0.55	0.20	0.10	0.10 0.40
7	33	0.33	0.09	0.15–0.60	0.35	0.13	0.10–0.60	0.15	0.12	0.00–0.45	0.17	0.11	0.05–0.40
8	31	0.35	0.09	0.10–0.55	0.39	0.12	0.15–0.55	0.19	0.13	0.00–0.50	0.17	0.09	0.10 0.35
9	33	0.34	0.06	0.20–0.55	0.36	0.10	0.20–0.55	0.25	0.12	0.00–0.40	0.18	0.13	0.05–0.35
10	27	0.40	0.10	0.20–0.60	0 40	0.15	0.10–0.70	0.13	0.12	0.00–0.40	0.15	0.10	0.05–0.25
11	19	0.33	0.08	0.20–0.50	0.40	0.13	0 20–0.70	0.19	0.12	0.05–0.40	0.20	0.08	0.10–0.30
12	15	0.35	0.13	0.20–0.65	0.39	0.05	0 15–0.60	0.17	0.12	0.10–0.35	0.13	0.07	0.05–0.20

Girls

Age	Number of Cases	T_1 Average	T_1 ± Standard Deviation	T_1 Range	T_2 Average	T_2 ± Standard Deviation	T_2 Range	T_3 Average	T_3 ± Standard Deviation	T_3 Range	Inverted T_3 Average	Inverted T_3 ± Standard Deviation	Inverted T_3 Range
3	..	....	....	.....	....	....	.....	....	....	.....	....	....	.....
4	22	0.34	0.07	0.15–0.50	0.30	0.09	0 10–0.40	0.07	0.09	0.00–0.20	0.14	0.07	0.05–0.30
5	33	0.34	0.10	0.20–0.55	0.32	0.14	0.10–0.60	0.13	0.10	0.00–0.30	0.13	0.07	0.05–0.30
6	29	0.27	0.11	0.10–0.50	0.30	0.12	0.00–0.55	0.09	0.08	0.00–0.30	0.10	0.02	0.05–0.15
7	35	0.30	0.09	0.10–0.40	0 32	0.10	0 20–0.60	0.13	0.09	0.00–0.30	0.11	0.03	0.05–0.20
8	30	0.29	0.10	0.15–0.50	0.34	0.12	0.10–0.60	0.11	0.10	0.00–0.30	0.14	0.08	0.05–0.30
9	26	0.30	0.12	0.10–0.60	0.37	0.11	0.10–0.55	0.13	0.09	0.00–0.30	0.12	0.06	0.05–0.25
10	18	0.29	0.09	0.10–0.50	0.31	0.13	0.10–0.60	0.14	0.11	0.00–0.30	0.13	0.05	0.05–0.10
11	21	0.30	0.11	0.10–0.50	0.26	0.12	0.00–0.50	0.15	0.12	0.00–0.45	0.12	0.07	0.05–0.30
12	9	0.31	0.10	0.10–0.50	0.36	0.12	0 20–0.50	0.17	0.07	0.00–0.30	0.08	0.02	0.05–0.10

was a slight average rise with years. A frequent form in lead II showed a fairly steep rise from the R-S complex with a blunt peak and an abrupt drop. In a high percentage of our cases, the T wave rose immediately from the R or S terminal at the base line with no S-T interval. It is interesting to note that in several of the records the origin of the T deflection was above the base line starting directly from the descending R limb, which in no case was raised more than 0.15 millivolts. According to Oppenheimer, an origin within 0.10 millivolts is normal in adults. Nicolai and Funaro, and Heubner noted a rapid increase in growth of the T wave with age, but most of their cases were in infants. Linetzky found a relatively small T deflection in young people, who had small hearts and low blood pressure.

The T deflection was not found inverted in the first or second leads in our series. In the third lead, the T wave was inverted in 175 of 497 records, or 35.5 per cent. An inverted T_3 preceded by an S wave occurred in 63 per cent of our cases. Seham found that 15 per cent of his subjects between the ages of 6 and 13 years showed T_3 inverted. Lewis found T_3 inverted fairly frequently in adults, although he said there was a tendency for the deflection to be upright. He found the inversion frequently when the Q-R-S complex was splintered.

VENTRICULAR PREPONDERANCE IN THE ELECTROCARDIOGRAM OF A NORMAL CHILD

There has always been some doubt as to how far hypertrophy of either the right or the left side of the heart can be identified in the electrocardiographic tracing. There is no question in regard to cases of marked hypertrophy as seen in such conditions as pulmonary stenosis, or mitral or aortic disease. Herman and Wilson [13] believed that preponderance was seen in the ventricular complex only when the ventricular weight was over 250 Gm. Lewis, in his study of fifty normal young adults, found several cardiograms which evidenced ventricular predominance on either the right or the left side when none was manifest on physical examinations. But in Lewis' [14] observations based on postmortem observations, he showed that Einthoven's [15] signs of ventricular predominance of the right and the left sides were better evidence of hypertrophy than physical observations.

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13. Herman, G., and Wilson, T.: Heart **9**: 9, 1921-1922.

14. Lewis, Thomas: Observations on Ventricular Hypertrophy, Heart **5**:367, 1913-1914.

15. Einthoven, H.: Weiteres über das Elektrokardiogram, Arch. f. d. ges. Physiol. **122**:517, 1908.

None of the eleven children who showed persistent enlargement of the heart in teleoroentgenograms showed any evidence of ventricular predominance in their electrocardiograms.

Three children showed a definite predominance of the right ventricle and eight showed predominance of the left, according to Einthoven's signs. The past history, physical examinations and teleoroentgenograms of these patients did not suggest cardiac hypertrophy in either ventricle, and in three cases the signs disappeared in later years. Some change in the arrangement of the ventricular or intraventricular conducting system was probably the reason for these variations in the tracings of normal children.

RECORDS IN FAMILY GROUPS

The characteristics which occur most frequently in family groups are those which differentiate the individual electrocardiograms. In some instances these are so similar in two related children that it is hard to distinguish one record from another. Resemblances were seen in seventy-five of the children in thirty-two families of our total group of forty families. The similarity was striking in many cases. In the families with three or more children, resemblances between electrocardiograms were more common than in the families with only two children.

A typical U wave may be present as a family characteristic, while splitting of the Q-R-S complex is frequently seen and notching of the R deflection may occur throughout a family group. Such a rarity as an inverted or aberrant P_3 also occurs as a family characteristic, and inverted or diphasic P's were found in four families, in one of which there were four children each presenting this peculiarity. Certain wave formations are frequently repeated in brothers and sisters. Especially is this true of the T waves, which often have similar points of origin in all members of a family. For example, in three members of one family the point of origin was elevated from the base line.

Just as many brothers and sisters resemble each other in some indefinable way, even though they may not have similar features, so most cardiograms of members of the same family bear a general resemblance to one another. This is the most common form of resemblance in the family group. High pointed deflections, or broad low waves with relatively slow transmission time, are common in family groups. In the work of one of us (G. N.) with children suffering from cardiac disease, the former type of wave has seemed to be associated more with nervous children and the latter type with phlegmatic ones. But in our series of normal children this relationship was not common.

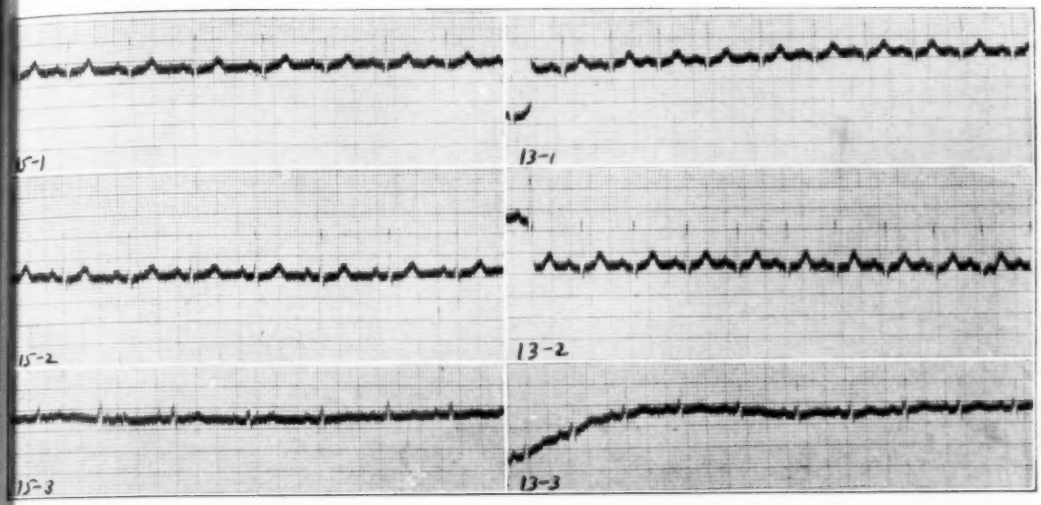

Chart 1.—Familial resemblances. *A* and *B*, electrocardiograms of two sisters showing marked similarity in the tracings, even to notched R waves in the third lead.

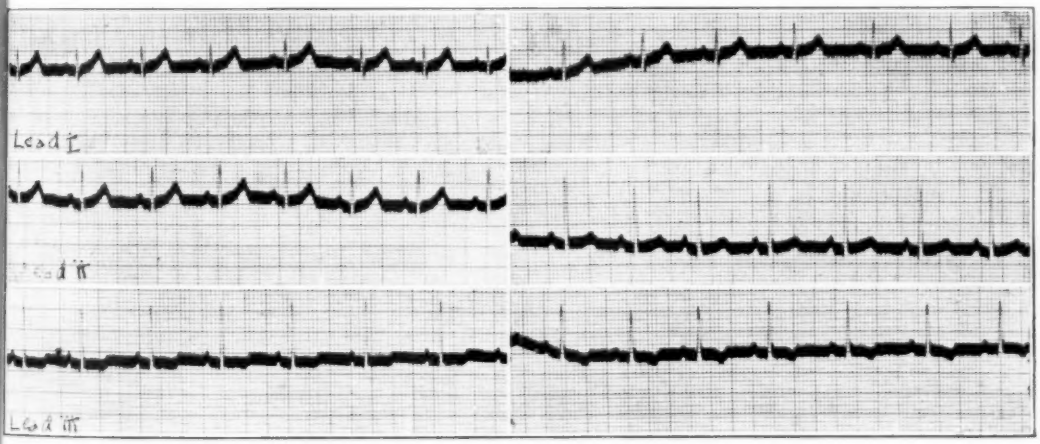

Chart 2.—Familial resemblances. *C* and *D*, electrocardiograms of a brother and sister, with marked likeness, especially in the first and third leads. The inverted T_3 in both tracings should be noted.

Uniformity of heart rate at the same ages appears to be another family characteristic. We have not been able to find in the literature any reference of familial resemblances in electrocardiograms.

THE ELECTROCARDIOGRAM IN SERIES

In following the changes made in a child's electrocardiographic records over a series of years, some interesting observations have been noted. Chief among these is the fact that the individual cardiogram in a child with a normal heart is always peculiar to that child. Characteristics that distinguish the record are retained with remarkable constancy throughout the series, which, in some instances, extended over seven years. Among the most prominent of these differentiating points

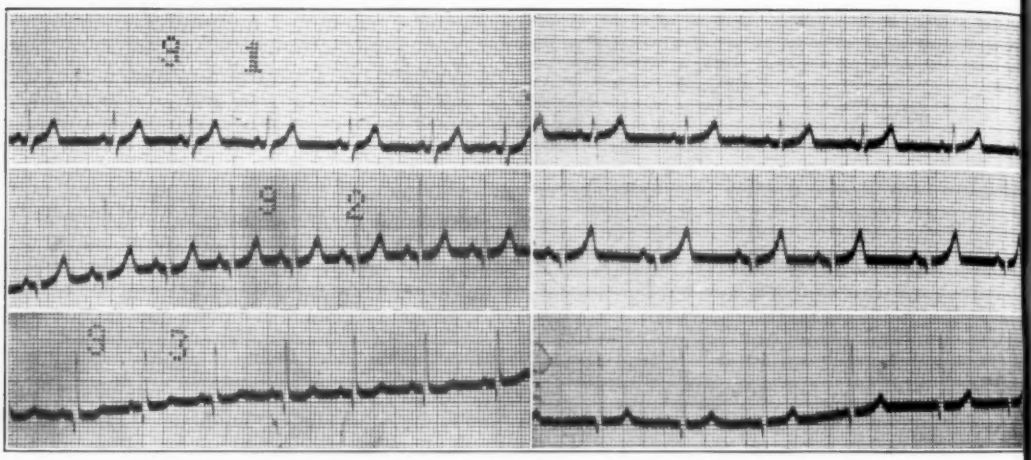

Chart 3.—Serial resemblances. *A*, electrocardiogram tracing taken at the age of 7 years. *B*, tracing of the same boy at the age of 13 years. The marked individuality of the tracing retained over a period of years should be noted; also, in *A*, the deeper S wave in the first lead and the more rapid pulse rate which are characteristic of the younger child.

which appear to be inherent qualities of individual cardiac activity are split Q-R-S complexes, inverted or diphasic P deflections and notching of certain waves. These qualities give individuality to the cardiographic record and are retained from one year to another.

In our series, there were sixty-three children who had from four to seven successive yearly cardiograms. The changes found in these series follow with fair regularity the average observations described for the various age groups. Thus, the pulse rate shows an almost constant decrease, while sinus arrhythmia increases in frequency. The Q deflections show most constantly a diminution in amplitude, as also do the S waves in the first and second leads. The T deflections are variable. On the whole, voltage increases with the series.

SEX DIFFERENTIATION IN THE CHILD'S ELECTROCARDIOGRAM

In our series of records we found that girls had a more rapid heart rate up to the age of 7 years and again at 12 years. Between these ages the boy's rate was greater. Arrhythmia was more marked in girls, suggesting greater vagal activity. The P wave in the second and third leads was higher in girls than in boys from the seventh to the eleventh years of age when the average heights were equal for the two sexes, becoming greater in boys than in girls at the age of 12 years. This is shown in chart 4. The Q waves, R_1, S_3 and the T waves

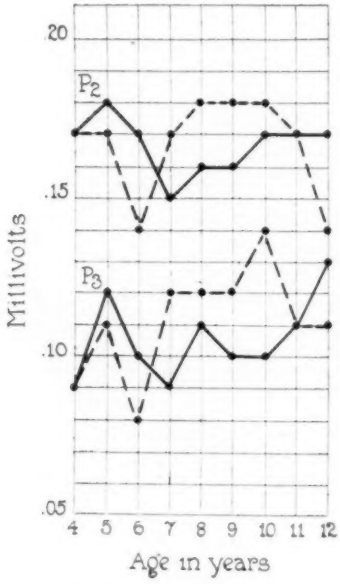

Chart 4.—Sex differences in the electrocardiograms of children. P waves in the second and third leads. The straight line represents boys; the broken line, girls.

were greater in boys than in girls at practically the same ages, and, as shown in charts 5, 6, 7 and 8, the Q and T waves were really greater at all ages. When the deflections in the boys' electrocardiograms were higher than those in the girls' records the difference was usually much more striking than when the reverse was true. All this may point to some difference between the auricles and ventricles in the sexes, at least between the age of 7 and 11. At the age of 12 years all the deflections of the electrocardiogram were higher in boys than in girls with the exception of T_3 in which the average wave is of equal height in boys and girls.

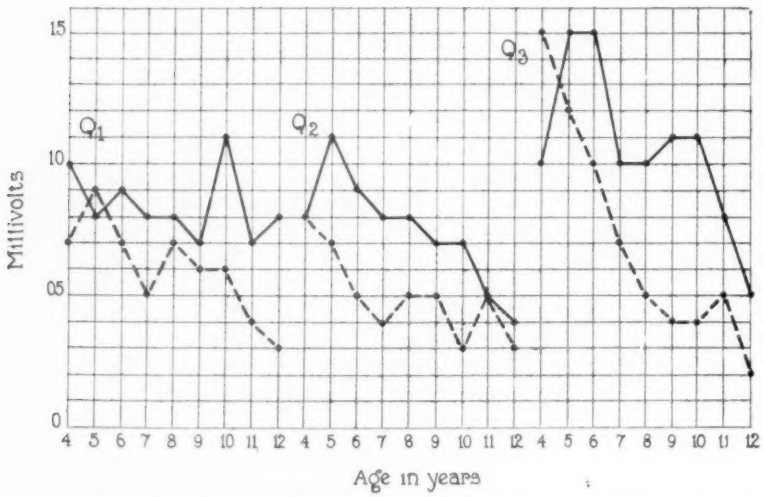

Chart 5.—Sex differences in the electrocardiograms of children. Q waves in all leads. The straight line represents boys; the broken line, girls.

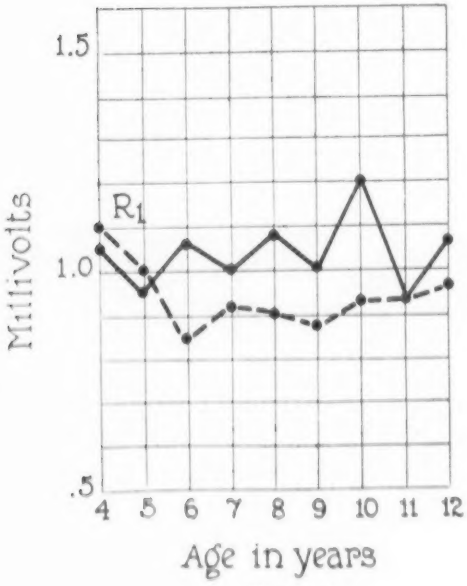

Figure 6

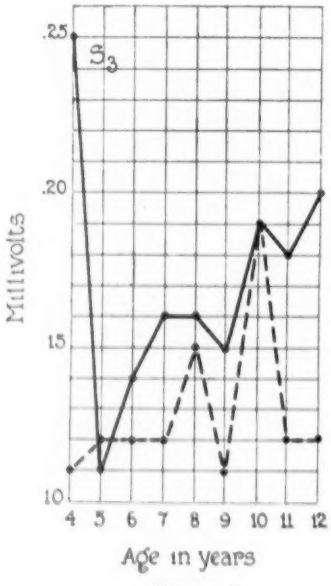

Figure 7

Chart 6.—Sex differences in the electrocardiograms of children. R wave in the first lead. The straight line represents boys; the broken line, girls.

Chart. 7.—Sex differences in the electrocardiograms of children. S wave in the third lead. The straight line represents boys; the broken line, girls.

In this connection it is interesting to note that Beneke,[16] in his work on the relationship between the upper and lower chambers of the heart and the sexes, found that the relative volume of the auricles to the ventricles was greater in the female than in the male.

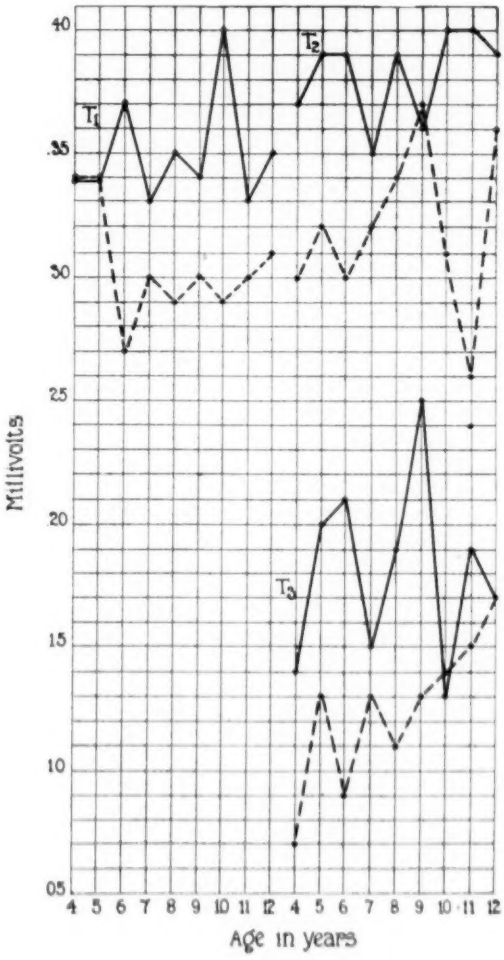

Chart 8.—Sex differences in the electrocardiograms of children. T waves in all leads. The straight line represents boys; the broken line, girls.

CONCLUSIONS

1. The electrocardiogram of a child shows a decrease in heart rate with increase in years.

2. Sinus arrhythmia increases in frequency as the child grows older.

16. Beneke, quoted in Tandler, J.: Anatomie des Herzens, ed. 3, Jena, Gustav Fischer, 1913, p. 122.

3. The transmission time is shorter in the heart of a child. This is seen both in the P-R and in the Q-R-S intervals. There is a slight increase in the length of the auriculoventricular interval after the seventh year.

4. The P deflection is higher in children than in young adults. P_3 was inverted in 10 per cent of our cases (3.2 per cent of these being constant in series), but such changes, present only in lead III, we do not regard as significant.

5. There is a decrease in the amplitude of the Q wave with the increase in the child's age, most marked in the third lead. Q and S are not constant in childhood.

6. The R deflection is much higher in the child than in the adult.

7. There is a tendency for the size of the S wave to decrease with the growth of the child except in the third lead in boys in which there is an irregular increase in depth from year to year.

8. Children have a broader and higher T deflection than is found in adults, and T frequently slopes directly from the R-S complex. There was an inversion of the T_3 in 35.5 per cent of our cases. The size of the inverted deflection decreases with the age period. We do not regard these changes in the third lead as of much significance.

9. Marked ventricular preponderance was present in only a few patients, none of whom showed clinical or roentgen-ray evidence of hypertrophy.

10. There is often a marked similarity in the electrocardiograms of members of the same family.

11. Characteristics that distinguish an electrocardiogram are retained with remarkable constancy throughout a series of years.

12. There is a sex differentiation in the electrocardiograms of children. The height of the P waves was greater in girls than in boys from the seventh to eleventh years of age, and the height of the Q waves, R_1, S_3 and the T waves was greater in boys than in girls at practically all ages studied. Sinus arrhythmia, when present, is more marked in girls than in boys.

PREVENTION AND MODIFICATION OF MEASLES WITH ANTIMEASLES DIPLOCOCCUS GOAT SERUM *

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During the past few years particular attention has been directed toward methods for the prevention of measles.[1] The need of satisfactory measures for accomplishing this purpose is frequently of vital importance, especially in children's hospitals or in the pediatric wards of general hospitals. When severely ill patients contract measles, any doubts pertaining to their recovery are multiplied.

Under any circumstances, measles is not merely a serious disease because of its possible complications, but one in which the mortality may be high, as shown by hospital reports.[2] Consequently, the advantage of providing immediate protection for an invalid child cannot be emphasized too strongly. One of us [3] (A. L. H.) has demonstrated the value of immune goat serum for purposes of this kind. Human convalescent measles serum should, no doubt, be equally effective, but a supply of this is not easily available, whereas, the possibility of producing immune goat serum in sufficient quantities to meet all requirements is a reasonable assumption. Furthermore, immunization of the horse with Tunnicliff's green-producing diplococcus has also been undertaken, and some results with the use of the serum thus produced have been reported.[4]

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* Received for publication, April 23, 1928.

* From the Children's Memorial Hospital, Chicago.

* The serum was supplied by Dr. Ruth Tunnicliff of the John McCormick Institute for Infectious Diseases, Chicago.

1. Weaver, G. H., and Crooks, T. T.: Further Observations on the Use of Convalescent Serum in the Prophylaxis of Measles, Wisconsin M. J. **23**:555 (March) 1925. Hoyne, A. L., and Gasul, B. M.: Measles Prophylaxis: Report on the Use of Immune Goat Serum, J. A. M. A. **87**:1185 (Oct. 9) 1926. Tunnicliff, R., and Hoyne, A. L.: The Prevention of Measles by Immune Goat Serum, J. A. M. A. **87**:2139 (Dec. 25) 1926. Hoyne, A. L.: The Prevention of Measles: Some Results with the Use of Tunnicliff's Immune Goat Serum, Illinois M. J. **53**:114 (Feb.) 1928. Tunnicliff, Ruth, and Hoyne, Archibald L.: Further Studies on a Diplococcus from Measles, J. Infect. Dis. **38**:48 (Jan.) 1926.

2. Emerson, H., and Hopping, A.: Scarlet Fever, Diphtheria and Measles at Willard Parker Hospital, New York City, Am. J. Pub. Health **15**:1026 (Feb.) 1925.

3. Tunnicliff and Hoyne (footnote 1, third reference).

4. Tunnicliff, Ruth, and White, Benjamin: An Anti-Measles Diplococcus Serum, Boston M. & S. J. **197**:272 (Aug. 18) 1927.

In considering the prevention of measles, whether by means of human convalescent measles serum, immune goat serum or the blood of adults who possess a past history of measles, the fact remains that the resultant protection is a passive immunity. Therefore, since the immunity is only transient, lasting perhaps from only three to four weeks, the recipient of the serum may be subjected to an attack of measles at some later date. With this thought in mind, we determined to administer immune goat serum to a series of patients regardless of the number of days which had elapsed since exposure. This was done, not with the idea of preventing the disease, for it has already been pointed out that for the latter intent serum should be given within three days of exposure, but with the object of ascertaining whether the serum would exert any influence on the course of the infection.

If immune goat serum administered during the incubative period of measles proved to lighten the course of the infection, the development of complications would not be anticipated to the same degree as in a severe attack of measles. Moreover, a patient treated in this manner would possess a permanent immunity following recovery from measles.

The work on which this report is based was begun in November, 1926, when an outbreak of measles occurred in the Children's Memorial Hospital. It had been our intention to extend the investigation over a much larger series of patients, but since we have been restrained from doing this through a lack of opportunities, we believe that, even with the limited number, the results are interesting.

Perhaps it should be unnecessary to state that serum was given only to patients possessing a definite negative history for measles. Between November, 1926, and April, 1927, all patients exposed to measles were divided into three groups. In the first group there were thirty-eight children ranging in age from 4 months to 12 years. There was only one child under 1 year of age, and only five who were more than 6 years old. Included in this group, there were also five affiliate nurses who professed never to have had measles. Nearly all of these contacts were immunized on either the first or second day of exposure, receiving 10 cc. of the immune goat serum intramuscularly. Measles did not develop in any one of the group. Among the forty-three subjects, two had a generalized urticaria from the serum, and four had slight local reactions.

In the second group, there were thirty-eight patients, the youngest being 18 months and the eldest 8 years of age. All but one patient received 10 cc. of immune goat serum. The time it was given varied from the fifth to the twentieth day following exposure to measles. Every one in this group developed measles. In some instances, the serum was not injected until the day before the rash appeared. We believe the course of the disease was modified in thirty-one of the

thirty-eight patients. However, it is true the infection might have been of a mild type in some of the thirty-one, regardless of the serum therapy. Only two of the thirty-eight children had any complications. One child, 2 years of age, developed otitis media on the right side, and the other, a boy, aged 4 years, had a bilateral otitis media. The latter patient received 5 cc. instead of the customary 10 cc. immune goat serum thirteen days after exposure to measles. There were no deaths and there was only one instance in which any reaction from the serum was noted. This reaction, moreover, was purely local and slight.

There were twenty-seven patients in the third group, their ages ranging from 1 to 11 years. All but six were 5 years old or younger. Serum was not administered to any of these; all of them developed measles. The attack was classed as severe in ten instances, average in fifteen and mild in two. Eleven of the group developed complications; ten patients had otitis media, three of which were bilateral. One of the ten patients with otitis media also had streptococcus meningitis which caused death. The eleventh patient with a complication had bronchopneumonia. One other child suffering from a severe attack of measles, but showing no complication during the acute stage, died three weeks later from a condition diagnosed as myocarditis.

SUMMARY AND CONCLUSIONS

1. Forty-three persons with a negative history for measles received immune goat serum within four days of exposure to measles and did not contract the disease.

2. Thirty-eight children exposed to measles were given immune goat serum five days or more after exposure, and all contracted the disease. In most instances (81.5 per cent), however, the disease appeared to be modified. Only two patients (5 per cent) had any complications, and none died.

3. Twenty-seven children who did not receive goat serum contracted measles. Eleven (40 per cent) developed complications and two (7 per cent) died.

4. On the basis of our observations, it seems that: (*a*) Tunnicliff's immune goat serum is of undoubted value as a prophylactic against measles when administered early, and (*b*) that it may exert a modifying influence on the course of the disease if given at any time during the incubative period.

25 E. Washington Street.

Case Reports

CONGENITAL CYST OF THE LUNG*

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AND

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MINNEAPOLIS

The American literature, until recently, has devoted little space to the subject of congenital cysts of the lung. Koontz,[1] in 1925, in an extensive review of the literature, did not find a single paper by an American author describing this congenital anomaly. His bibliography showed that the largest number of cases was reported by the Germans and a few by the English, French and Italians. In the last two years, papers have been published by Miller[2] and Moore,[3] Americans, and by Smith[4] and de Lange,[5] foreign authors. The total number of reports occurring in readily accessible literature at the present time amounts to 112.

Briefly reviewing the summary of Koontz' data, one finds that there is no predilection to this condition in either sex. In twenty-six of his 108 cases, the right lung was affected; in forty-six, the left, and in twenty-seven cases, both lungs were involved. In a few of the cases reported with involvement of the right lung, there was a history of symptoms identical with those in the case to be reported.

REPORT OF CASE

Our case was that of an infant girl referred to the hospital for treatment of malnutrition as well as abnormal breathing. Before she entered the hospital, the history was as follows: The mother had on several occasions called the attention of the nurses and her visiting physician to the abnormal type of breathing of her infant during the puerperium. After the mother left the hospital, she had fre-

* Received for publication, April 23, 1928.

* From the Department of Pediatrics, University of Minnesota.

1. Koontz, Amos R.: Congenital Cysts of the Lung, Bull. Johns Hopkins Hosp. **37**:340, 1925.

2. Miller, R. T.: Congenital Cystic Lung, Arch. Surg. **12**:392 (pt. 2) (Jan.) 1926.

3. Moore, W. F.: Multiple Cysts of the Lung, Ann. Otol. Rhin. & Laryng. **36**:263, 1927.

4. Smith, S.: Congenital Cystic Disease of the Lung, Brit. M. J. **1**:1005, 1925.

5. de Lange, C.: Congenital Cystic Lung and Agenetic Bronchiectasia, Nederl. Tijdschr. v. Geneesk. **1**:2515, 1926.

quently observed attacks of cyanosis coming on, apparently after the least bit of exertion on the part of the child.

When the infant was 7 weeks old, a careful physical examination revealed that she was frail, and was breathing at the rate of from 60 to 70 a minute. The breathing was of an obstructive type, with moderate retraction in the supraclavicular and epigastric regions. There was impaired resonance over the right side of the chest, anteriorly and posteriorly, particularly in the midaxillary line. In this area there was also suppression of breath sounds. The left side of the chest presented signs of a compensatory emphysema. The temperature was normal; a leukocyte count was 10,400; the hemoglobin was 95 per cent, and there was a moderate cyanosis.

Fig. 1.—There is a dense opacity throughout the whole right hemithorax with some areas of clearness near the base at the periphery. There is a marked displacement of the heart and mediastinal structures to the left side.

A thoracentesis done at this time yielded from 40 to 50 cc. of a limpid, viscid fluid, comparable to the white of egg. This fluid was analyzed and found to be free from fat and blood, but it contained a large amount of albumin. The thoracentesis seemed to make the child more comfortable. After an interval of six days it became necessary to repeat the tapping, and this procedure was continued until admission. The roentgenographic appearance at this time is shown by figures 1 and 2.

At the time of entrance to the University Hospital, the child was 10 weeks old and weighed 8 pounds and 13 ounces (3,995 Gm.) (the weight at birth was 6 pounds

[2,725 Gm.]). The hemoglobin was 63; the erythrocytes numbered 4,090,000 and the leukocytes, 10,700. The urine was normal. The infant did not have fever. Respirations were 30 per minute. There was no cyanosis and apparently no dyspnea. The mother reported that when the child's lung filled up, she nursed poorly and finally refused food. Two days after admission, 180 cc. was aspirated from the right thoracic cavity. The appearance of this fluid was the same as that described, except toward the end of aspiration, when the fluid became dark colored as if mixed with old blood. A microscopic examination of the fluid showed that it was contaminated and that it contained both red and white blood cells. Three days after the first aspiration, a second 180 cc. of fluid was removed, and three days later, 200 cc. more. Figures 3, 4 and 5 show the cyst before and after the injection of sodium iodide.

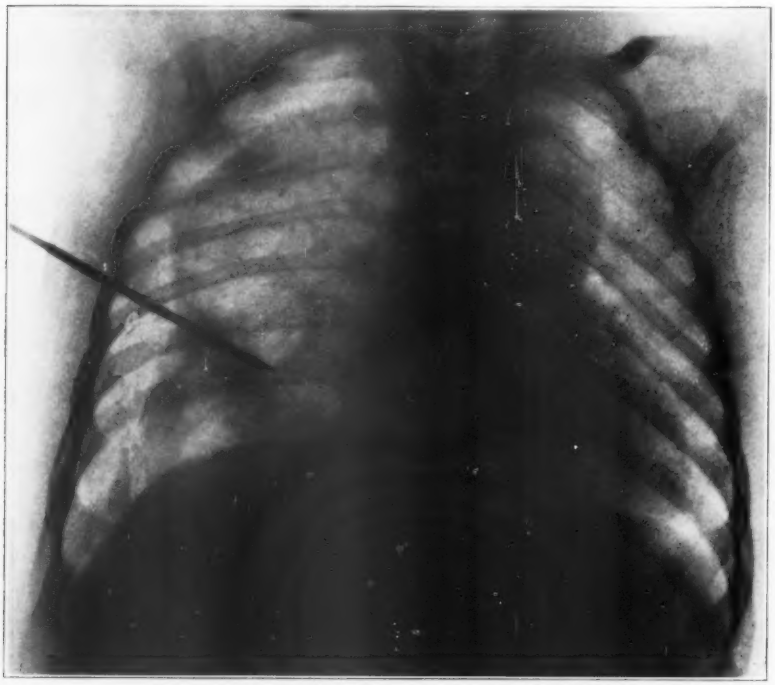

Fig. 2.—The needle is shown in the partially collapsed lung after withdrawal of fluid. There is a large amount of air in the pleural space. Areas of rarefaction may be seen in the collapsed lung.

The continuous aspiration of this closed cavity was beginning to be a serious problem. The rapid filling was disturbing the nutrition so much that it was thought advisable to treat the child for the condition in the chest as empyema and to establish drainage. The surgical staff thought that the best way to accomplish this drainage was first to produce adhesions between the parietal and visceral pleura, and then to introduce a permanent drain by means of a trochar. The first stage of this operation was done under local anesthesia. An inch of each of the fourth and fifth ribs in the midaxillary line was removed and the area packed with iodoform gauze. Six days later, a trochar was introduced and permanent drainage established.

During the time of the minor operations and for several weeks after, the child's condition was poor. Frequent subcutaneous injections of saline solution and several blood transfusions of 100 cc. each were given. The weight dropped from 8 pounds and 12 ounces (3,965 Gm.) to 7 pounds and 14 ounces (3,575 Gm.) and the child by this time was almost 3½ months old.

The cavity was now aspirated twice a day, and about 50 cc. obtained at each aspiration. It was then suggested by the surgical staff that surgical solution of chlorinated soda (Dakin's solution) be introduced into the cavity after aspiration and left in for twenty minutes and then removed and followed by saline solution. This procedure seemed to increase the amount of cystic fluid secreted. The

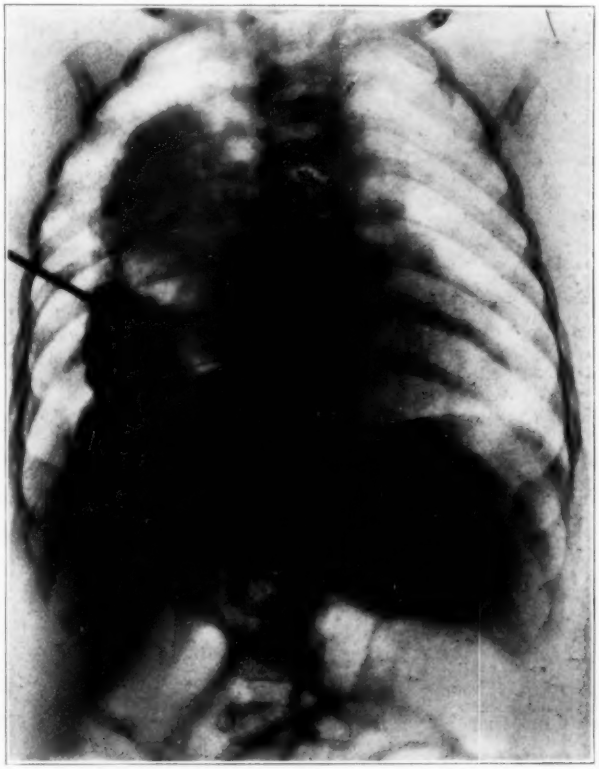

Fig. 3.—The needle is shown in the partially collapsed lung. Five cubic centimeters of sodium iodide was introduced into the cystic area. Some of the sodium iodide is clearly shown below the dome of the diaphragm.

irrigating solution was then changed to ½ of 1 per cent solution of formaldehyde in physiologic sodium chloride solution. About 20 cc. of this solution was used after each aspiration twice a day and appeared to be more irritating than the surgical solution of chlorinated soda. It was left in for only two to five minutes and then washed out with physiologic sodium chloride solution. However, the cavity became less and less sensitive, so that finally a 1 per cent solution of formaldehyde was used. It was hoped that this solution would destroy the secreting membrane. After about two months' treatment, it was found that the cavity gradually had

decreased in size, and would hold only from 5 to 8 cc. The decrease in size of cavity may be seen in figure 6.

It is significant to note that, in spite of these unusual therapeutic measures, the infant's weight increased during its stay of five and a half months in the hospital from 8 pounds and 12 ounces to 12 pounds and 2 ounces (5,500 Gm.).

For some time, bronchoscopy and injection of iodized oil 40 per cent had been under consideration. It was thought that by this procedure one might determine what relation, if any, the cyst had to one or more bronchi; also, whether smaller cysts might not be present. Unfortunately, the infant did not do well after this procedure. It lived about forty hours after the bronchoscopy. Figure 7 shows the distribution of iodized oil in the right lung.

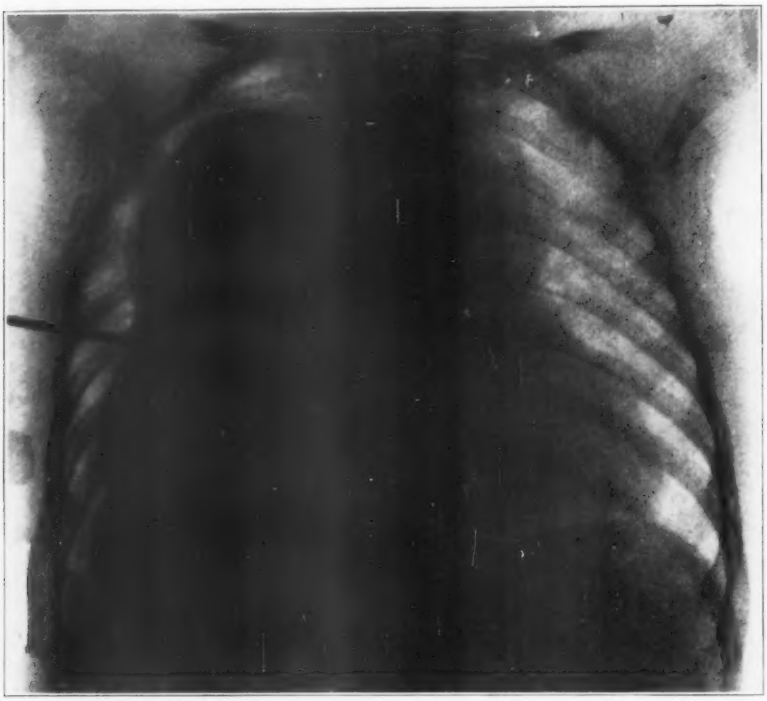

Fig. 4.—Approximately 60 cc. of sodium iodide was injected into the cyst. A clear outline of the whole lung and its various lobes are made out. None of the solution passed into the pleural space, and there was no evidence of any connection between the lung and esophagus or any other structure in the mediastinum.

The autopsy was limited to the chest. The cyst was removed for microscopic study. The entire right lung was functioning, except the part of the right lower lobe that contained the cyst. The posterior part of this lobe was adherent to the wall of the chest, and an especially strong adhesion had formed about the sinus. The gross dimensions of the cyst were: inside diameter, 1 cm.; length, 3 cm., and thickness of the wall, from 0.5 to 1 cm. The inside had a beefy appearance and there was no evidence of a lining membrane. A connection between the cyst and a bronchus could not be demonstrated. Serial sections were not made.

Microscopic sections from the cyst wall showed the presence of mucous glands, a layer of submucosa and a large amount of fibrous tissue in which osteogenetic tissue was invading cartilage. A thick layer of circular and longitudinal smooth muscle was also present. The pathologists diagnosed this condition as an enormously hypertrophied bronchus.

COMMENT

Before the first roentgenogram was taken, atelectasis had been considered as a possible cause, but the duration of the symptoms and the finding of the mediastinal structures displaced to the left made this diag-

Fig. 5.—This lateral view indicates that the sodium iodide occupies the entire anteriorposterior thickness.

nosis practically impossible. In the differential diagnosis were considered a neoplasm, diaphragmatic hernia, bronchial cyst, chylothorax and maldevelopment in the lung, involving the blood or the lymphatic system. A barium meal was given which ruled out the hernia, and after the thoracentesis, it was suspected that we were dealing with a cyst. The introduction of sodium iodide established this diagnosis and in addition demonstrated the fact that it was intrapulmonary.

Fig. 6.—The cyst is shown filled with sodium iodide and with the catheter in position. This picture shows the marked decrease in size of the cyst after weeks of treatment.

Fig. 7.—There is considerable filling of the alveoli of the lung with iodized oil 40 per cent, indicating that the lung is fairly well aerated at this time. The cyst is shown by a small clear area about the catheter. A definite connection does not appear to exist between the bronchi and this cyst.

The genesis of this type of deformity of the lung has not been clearly explained by embryologists or pathologists. Heuter [6] believed that cysts develop because of stenosis of bronchial branches. The earlier this abnormal process develops, the fewer and larger the cysts. The presence of mucous cells, smooth muscle and cartilagenous tissue classifies this cyst as of bronchial origin. Had we not used solution of formaldehyde, more confirmatory evidence might have been obtained from the presence of a lining membrane.

Miller [2] was of the opinion that these cysts are not caused by a retention of secretion resulting from strictures. "This," he said, "can hardly be the actual cause; since a communication with the bronchus has been demonstrated frequently and the epithelium is not always flattened; since there is no apparent presence of atrophy of the intervening septums, and the secretion found in the cavity is not sufficiently great in amount to cause a distention." The case under consideration contradicts some of these assumptions. The secretions into a cyst can cause marked distention and distressing symptoms. Atrophy of the intervening septum and smooth muscle may not occur even though the cyst is large and has existed for a long time. The stricture or stenosis of a bronchus or bronchiole is probably due to developmental error and, if so, precludes obvious genetic explanation.

CONCLUSION

A description of a single congenital cyst of the right lung is presented. The microscopic study indicates that it originated from a bronchus.

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6. Heuter, C.: Ueber angeborene Bronchiektasein und angeborene Wabenlunge, Beitr. a. path. Anat. u. z. allg. Pathol. **59**:520, 1914.

GRANULOCYTIC APLASIA OF THE BONE MARROW FOLLOWING THE USE OF ARSENIC*

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Arsenic poisoning in children has been reported only occasionally. Veeder and Jeans [1] referred to three cases seen in the course of several thousand injections of arsphenamine. In one case the liver was affected and there was severe jaundice; in another there were skin disturbances; in the third, which was fatal, there was found an encephalitis.

McBride [2] reported a case of "hemorrhagic encephalitis" in a boy, aged 4, which resulted from the administration of arsphenamine. He noted increased pulse rate, headache, nausea, general convulsions, pain in the extremities, twitching of the hands and feet, deep coma and abdominal distention. Following the intravenous administration of sodium thiosulphate, the boy recovered.

Dodd and Wilkinson [3] have recently reported the case of a colored girl, aged 11, with severe granulocytic aplasia of the bone marrow following the administration of sulpharsphenamine. The symptoms in their patient were pain in the side; enlarged liver and spleen; general glandular enlargement; temperature ranging from 101 to 105 F.; foul, painful mouth and throat; redness of the palms and soles; leukopenia varying from 4,500 to 136 with absolute disappearance of polymorphonuclear neutrophils; red blood cell count of 4,850,000; 80 per cent hemoglobin, and diminished platelet count. The child died. Autopsy revealed almost complete aplasia of the bone marrow, bronchopneumonia, and enlargement of the liver without definite pathologic conditions.

In addition to the case reported, Dodd and Wilkinson [3] collected from the literature twenty-three cases of arsenic poisoning in adults following the administration of organic arsenical compounds. Particular stress was laid on the blood changes. Nine of these patients had severe anemia, ten had moderate anemia, two had slight anemia, and two showed no anemia. The white blood count varied from 500 to 8,500 (two cases were reported with 29,000 and 76,000, respectively). Polymorphonuclear neutrophils varied from 0 to 88 per cent. Platelets varied from 10,000 to 180,000 per cubic centimeter. Hemorrhages were present in nineteen

* Received for publication, April 19, 1928.

* From the Milwaukee Children's Hospital.

1. Veeder and Jeans, in Abt: Pediatrics, **5**:656, 1924.

2. McBride, R. H.: Treatment of Arsphenamine "Encephalitis" with Sodium Thiosulphate, J. A. M. A. **84**:729 (March 7) 1925.

3. Dodd and Wilkinson: Severe Granulocytic Aplasia of the Bone Marrow, J. A. M. A. **90**:663 (March 3) 1928.

of the twenty-three cases. There was involvement of the mouth and pharynx in ten cases. Death occurred in thirteen instances, and recovery in ten. Postmortem examination in nine instances revealed hemorrhages and aplastic bone marrow. In every case in which the patient recovered the white blood count was 2,700 or above, and in these the polymorphonuclear neutrophil percentage fell below 34 in only three cases (10, 7 and 0 per cent).

The case here presented is of interest because of the following factors: sudden onset following the prolonged use of potassium arsenite, high fever, enlarged liver and spleen, lesions of the mouth, and, particularly, the changes in the blood picture with subsequent recovery.

REPORT OF A CASE

M. E. S., a girl, aged 9, was admitted to the Milwaukee Children's Hospital in the Medical service of Drs. A. L. Kastner and J. C. Zuercher on June 28, 1927. The child was one of twins, being born at full term with normal delivery. Her weight at birth was 5 pounds (2.3 Kg.). She was breast fed for three months following which she was bottle fed with the ordinary milk mixtures; she did not make satisfactory progress. She was not given orange juice, cod liver oil or vegetables. She sat up at 18 months, walked at 3 years, and was always weak. Muscular twitchings had been present since she was 1 month of age. There was a history of measles, whooping cough and tonsillitis. The tonsils and adenoids were removed in October, 1926. Physical examination on admission revealed four carious teeth and a moderately inflamed pharynx, with no cervical glandular adenopathy. The heart and lungs were normal. The liver and spleen were not palpable. There was generalized involuntary muscular twitchings. The temperature was 102.4 F.; the pulse rate, 120 and respiration 20. The white blood cell count was 14,400 with 62 per cent polymorphonuclears, 34 per cent lymphocytes, 3 per cent large mononuclears, and 1 per cent transitionals. The red count was 4.77 millions and the hemoglobin 80 per cent. The urine was normal. Cultures from the nose and throat showed a mixed infection of staphylococci and streptococci. A diagnosis of cerebral injury at birth with athetosis and acute pharyngitis was made. The pharyngitis quickly subsided, but the involuntary muscular twitchings failed to improve under treatment with phenobarbital and acetylsalicylic acid. Mantoux intracutaneous tuberculin tests were negative in dilutions of 0.01 and 0.1 mg., as were the Wassermann and the microprecipitation tests for syphilis.[4]

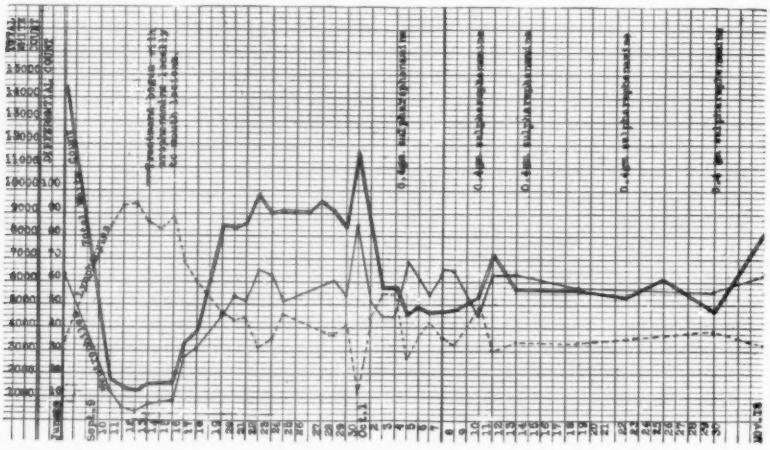
Treatment with phenobarbital, 1.5 grains (0.09 Gm.) daily was begun on August 8, in conjunction with potassium arsenite. The dosage of the potassium arsenite was started at 6 minims daily, and was increased 1 minim daily until September 6, when 36 minims were given. On this day the urine showed one plus albumin, and the involuntary muscular movements were markedly increased. The potassium arsenite was discontinued after the morning dose on September 7. A total of 625 minims of potassium arsenite, which corresponds to 0.41 Gm. (6 grains) of arsenic trioxide, had been administered within thirty days.

On September 9, the child's temperature at 8 o'clock in the morning was 99 F. At 10 o'clock, she attempted to walk. At 1 o'clock in the afternoon, she was irritable

4. Peterman, M. G.: Microprecipitation Test for Syphilis, Am. J. Dis. Child. **34**:404 (Sept.) 1927.

and complained of noises about her. At 4 o'clock, the temperature was 104.2 F., with a pulse rate of 140, and respiration 40. The child complained of headache. Examination revealed a flushed face, a slightly inflamed pharynx, gingivitis and infected teeth.

On the second day, the temperature rose to 105 F., and the child still complained of headache. The tongue was coated; the breath was fetid; and there was moderate inflammation of the anterior faucial pillars, but there was no membrane. The gums were reddened and moderately swollen. The roots of the second upper left deciduous molar were present in the gum, above which there was an ulcerating area covered with foul, purulent matter. The organisms of Vincent's angina were recovered from this ulcer in great numbers, and elsewhere in the oral cavity. Throat cultures showed a mixed infection of staphylococci and streptococci. A group of soft, painful submaxillary glands were present. The heart and lungs were normal; the liver was palpable 7.5 cm., and the spleen, 5 cm., below the costal margin. The abdomen was distended and the child



Relationship of lymphocytes and neutrophils to total white count.

was constipated. There was no tenderness, rigidity or jaundice. The white blood count was 1,800 (repeated and checked) with lymphocytes, 84 per cent; large mononuclears, 4 per cent, and neutrophils 12 per cent. The blood pressure was systolic, 96; diastolic, 68. The results of the ophthalmoscopic examination were negative. The urine gave a two plus reaction to the test for albumin, with a negative microscopic examination. The white blood cells gradually decreased in total count, with a relative decrease of neutrophils and a relative increase of lymphocytes, until on the fourth day of fever, (104.6 F.) the white count was 1,300 with 93 per cent lymphocytes, 4 per cent large mononuclears and only 3 per cent neutrophils. Abnormal types of white cells were not observed with Wright's and peroxidase stains. The red cell count was 3.17 millions, with the hemoglobin, 48 per cent, and with marked anisocytosis and poikilocytosis. The blood platelet count was 216,000. The blood calcium was 7.44 mg.; phosphorus, 1.4 mg.; sugar, 87 mg.; urea, 4 mg.; urea nitrogen, 18 mg.; creatinine, 1.1 mg., and nonprotein nitrogen, 21.6 mg. Successive blood cultures were negative for forty-eight hours, as were Wassermann tests of the blood, and microprecipitation

tests. Blood sedimentation time[5] was slightly increased, being 9 mm. for one-half hour; 24 mm. for one hour; 53 mm. for two hours, and 108 mm. for twenty-four hours. The Widal test of the blood was negative. The temperature gradually subsided following institution of treatment for Vincent's angina; namely, alternate applications to the gums of arsphenamine in glycerine, and 10 per cent hydrogen peroxide. The spleen was no longer palpable on the seventh day, when the temperature became normal, yet the liver remained palpable 4 cm. below the costal margin. The submaxillary glands were no longer palpable on the eleventh day, at which time the liver was palpable 1 cm. below the costal margin.

No arsenic was revealed in the urine by Reinsch's and Gutzeit's tests on September 21, fourteen days after the potassium arsenite had been discontinued. The lesions of the mouth first healed following local treatment, and there was a more gradual return of the blood picture to normal. Mouth smears on November 3 showed Vincent's organisms. Sulpharsphenamine, 0.4 Gm., was injected at weekly intervals intramuscularly for five weeks without disappearance of the organisms of Vincent's angina, and without any change in the clinical or blood picture. On November 18, the white blood count was 7,800 with 35 per cent lymphocytes, 3 per cent large mononuclears and 62 per cent neutrophils. Mouth smears made on the same date showed Vincent's organisms.

In this case the white blood count dropped from 14,400 to 1,300 cells, with a decrease in the polymorphonuclear neutrophils from 62 to 3 per cent, and a corresponding increase in the lymphocytes from 34 to 93 per cent. As shown in the chart, the total white blood count gradually rose again, with progressive increase in polymorphonuclear neutrophils and relative decrease in lymphocytes. On the seventh day following the onset of fever, the temperature became normal. The white cell count was 8,450 with 56 per cent lymphocytes, 2 per cent large mononuclears and 52 per cent neutrophils. The clinical picture showed marked improvement with increase in neutrophils, and an actual increase in total white cells. At no time were jaundice, rose spots or generalized adenopathy present. Two weeks following the onset of the fever, complete recovery from all symptoms had taken place, with the exception that the organisms of Vincent's angina were still obtained in small numbers from the mucous membranes of the mouth. The white count was 9,050, with 46 per cent lymphocytes, 51 per cent polymorphonuclear neutrophils, 1 per cent transitionals and 2 per cent large mononuclears. The child's athetosis was much improved following this attack. Whether this was due to the fever, comparable to the inoculation of persons with general paralysis with malarial organisms to produce high fevers, or whether other factors brought about this change, can only be conjectured.

Ulceration of the mucous membranes took place simultaneously with the decrease of polymorphonuclear neutrophils. This can be explained on the basis of the lowered general body resistance as manifested by the decrease in granulocytic cells. Secondary infection, such as Vincent's angina in this case, or the lesions of the mouth and pneumonia in Dodd and Wilkinson's series, becomes thus easily established.

Apparently this is the first case on record with marked changes in the blood picture due to inorganic arsenic as such. In Dodd and Wilkinson's series of cases, the changes were caused by the administration of

5. Westergren: Die Senkungsreaktion, Ergebn. d. Inn. Med. u. Kinderh. **26:** 577, 1924.

arsphenamines, which are organic arsenical compounds. Cushny [6] stated that arsphenamine "is found in the urine for some days, apparently in the form of arsenites or arsenates" which are inorganic arsenical compounds. He also stated that arsenic is stored in the liver and bone marrow, and is excreted in the urine, feces, breast milk and skin, and that the excretion of neoarsphenamine resembles that of arsphenamine except that formaldehyde appears in the urine from the decomposition of the molecule. The picture here described is then due essentially to the same process, whether the arsenic is administered in the organic or inorganic forms.

The facts that arsenic is stored in the body in the bone marrow for some time, and that autopsies on patients dying with this condition have revealed marked aplasia of the bone marrow, especially in regard to the granulocytic elements of the blood, would point to the destructive action of arsenic on these blood elements. In this instance, following the daily administration of potassium arsenite, the arsenic reached a concentration in the body sufficient to cause granulocytic aplasia of the bone marrow. Sulpharsphenamine, administered at weekly intervals following complete recovery from all symptoms due to the potassium arsenite, did not produce any reaction, in all probability due to the lack of concentration of the inorganic arsenic end-products in the body tissues.

Attention is called to the marked similarity of the picture of this type of arsenic poisoning to that of agranulocytic angina. The differentiation may be difficult. The etiology of agranulocytic angina is unknown. Zadek,[7] Schultz and Jacobowitz [8] in three instances removed bone marrow from the sternum during the height of the disease, and found a cell-poor marrow with decrease in the granulocytic elements. They found the same changes at autopsy and concluded that there was a primary injury to the leukomyeloid apparatus. Moore and Wieder [9] expressed the belief that there is a toxin circulating in the blood stream, having an affinity for, and spending its force on, the neutrophil producing structure of the bone marrow. Could not this unknown toxin in some cases be arsenic? Indeed, Dodd and Wilkinson question if a case reported by Kastlin [10] of Toronto as agranulocytic angina is not arsenical poisoning, since the disease began after two years of arsenical treatment for syphilis, and followed soon after an injection of neoarsphenamine.

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6. Cushny: Pharmacology and Therapeutics, ed. 8, 1924, p. 610.

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In making a diagnosis it is necessary to differentiate this condition from acute poisoning due to thorium, benzene and roentgen rays, and such conditions as agranulocytic angina, acute leukemia, aleukemic leukemia and severe sepsis. This patient had not been subjected to treatment with thorium compounds, benzene or roentgen rays. In acute leukemia, there is a tendency to hemorrhage, and the leukocyte count is usually over 50,000 with abnormal types of lymphocytes present. In this case, there was no hemorrhage, and the lymphocytes were mature and normal, as demonstrated by peroxidase and Wright's stains. In aleukemic leukemia, immature and abnormal lymphocytes are present, and the blood picture tends to approach that of leukemia. In severe sepsis, there are usually foci of infection to explain the severity of the symptoms, or septicemia. In infectious mononucleosis [11] there is usually a leukocytosis rather than a leukopenia, and abnormal types of cells are found in the blood. The negative Widal test and the blood culture and the absence of clinical symptoms ruled out typhoid fever. Agranulocytic angina presents the same blood picture, but occurs in four fifths of the cases in women over 40, and the outcome has been almost invariably fatal.

SUMMARY

Six hundred twenty-five minims of potassium arsenite (0.41 Gm. or 6 grains of arsenic trioxide) produced granulocytic aplasia of the bone marrow in a child, aged 9.

167 Seventeenth Street.

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SHOE-DYE POISONING

REPORT OF THREE CASES *

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A brief clinical report of three cases of shoe-dye poisoning is presented in order to call attention to this form of poisoning, to which children are particularly susceptible.

While from 1900 to 1926 sixty-one cases were reported in the literature, little has appeared in journals of pediatrics, although it is a condition that pediatricians may be called on to diagnose.

The outstanding symptom is general cyanosis, which is as marked as in congenital cardiac disease. This begins from one-half to several hours after the shoes are put on. It is accompanied by general weakness, chills, headache, vertigo, somnolence, nausea and occasionally by stupor. Prostration is present in severe cases. The pulse rate is rapid.

Unless this form of poisoning is borne in mind, diagnosis is impossible, and the cyanosis remains a serious and unexplained problem to the physician and parents.

Poisoning is produced by shoe dye applied to tan shoes to produce a black or brown. The poison is absorbed through the intact skin and little if any discoloration may result. There may or may not be a strong odor to the dye. The poisoning is more apt to occur if the shoes are damp, especially if exercise induces perspiration.

If the shoes are removed, the cyanosis begins to decrease but may still be present twelve hours later, as in one case here reported.

Probably most of the cases are mild and so transitory that they are missed. As Muehlberger showed, the diagnosis is often missed by the physician. While the condition is usually of only a few hours' duration, two deaths are reported.

Several cases of nitrobenzene and aniline poisoning in infants have been reported, due to the ink used in marking diapers.

The toxic agent in the shoe dye is not entirely clear. It has been claimed to be aniline, nitrobenzene, impurities in aniline, tetranitromethane or the pigment in the dye. After a careful study, Muehlberger concluded that it is due to either aniline or nitrobenzene used as a solvent for the dye. The later cases in this country have been caused by aniline.

It is strange that either solvent would cause nearly identical symptoms but both are benzene derivatives of the coal tar series, aniline

* Received for publication, Feb. 1, 1928.

being reduced from nitrobenzene. Many cases of industrial poisoning are reported from these and other related chemicals.

The dye was not analyzed in my cases.

Little laboratory work is published in any of the reports. The blood counts are normal, and in one case the urine contained a trace of albumin. The exact change in the blood is not given, for while Sanders reported methemoglobinemia, Muehlberger did not find it in aniline poisoning.

REPORT OF CASES

CASE 1.—A. M. D., a boy, aged 6 years, was seen on March 24, 1926, with Dr. Waffle. Ten days before, while in a store with the mother, he became blue about the mouth and was dizzy, but did not have pain. The cyanosis became more marked before the mother could get home. He became unconscious; the body was rigid and the teeth set. Dr. Waffle was called and thought the child about to die. When the child was put in bed he gradually improved, but when he was up and about the next day he had another spell and became blue but not unconscious. This lasted several hours. The child has been somewhat weak since that time.

The boy had had diphtheria at the age of 3 months. He had had several attacks of bronchitis and tonsillitis. He had not had convulsions or cyanosis previously. The consultation was called to determine the cause of the attacks.

Questioning brought out the fact that the child had worn shoes that had been dyed four days before. The shoes were put on about 11:30 a. m. and the cyanosis began about 2:40 p. m. The cyanosis improved when the shoes were removed, only to return the next day when the shoes were worn. Nothing was found to explain the condition on physical examination.

While epilipsy was considered, a diagnosis of shoe-dye poisoning was made from the history. Several weeks afterward the child had a somewhat milder spell, and it was found by the mother that the child had two hours before discovered the shoes in the attic and had put them on. The cyanosis cleared up when the shoes were removed. Further attacks did not occur.

CASE 2.—R. G., a boy, aged 13 years, was seen on March 20, 1926. The night before, he came home and fell on the floor from weakness. He was at once put to bed. In the morning marked general cyanosis was noticed, and he complained of severe headache.

This boy had been under my care since his infancy and had never had a similar attack nor any illness bearing on the case. He had as marked cyanosis as I have ever seen, but otherwise did not seem seriously ill. His heart was normal and he did not have a temperature. The diagnosis was quickly cleared up when it was learned that he had on the previous evening worn shoes that had been dyed the day before. The hemoglobin was 80 per cent and the blood count normal. The urine was normal and contained a trace of albumin. There was a slight odor of shoe dye from his feet but no discoloration. The stockings and shoes had a strong odor of shoe dye.

He was given a bath, and after three or four hours the cyanosis faded. The headache cleared up with the cyanosis, but the boy was weak for several days. He has not had cyanosis since that time.

CASE 3.—G. H., a boy, aged 9½ years, had tuberculous cervical adentitis two years before I saw him. The glands were removed at the Rochester General

Hospital, and he made a good recovery. The family history and previous history were unimportant. There had not been any similar trouble in the family.

He was brought to my office on July 26, 1927. One week before, he became blue. This was noticed by his family and by the neighbors. He did not have headache but showed a marked lassitude and remained about his home all day.

The condition cleared up, but after the attack the mother became more anxious to learn the cause and to know if it was likely to return. His shoes had been dyed black the morning before the attack. He put them on at 8:30 a. m. and the cyanosis began two hours later. The shoes were worn until 7 p. m. It was a hot day, and the boy perspired considerably. The shoes were worn again four days later without ill effects.

Directions were given on packages of dye to avoid the use of shoes for twenty-four hours after dyeing. That this is not sufficient time for children with marked susceptibility is shown by the first case reported. The latest packages of dye give seventy-two hours as the limit.

Dr. Guldin of this city had a patient, a girl, aged 9 years, who showed a return of the poisoning when the shoes were worn nine days after the first attack.

TREATMENT AND PROPHYLAXIS

Either the use of aniline and nitrobenzene should be prohibited or the public, boot blacks especially, should be warned of the danger of poisoning. Many men doing this work have little knowledge of English, which adds to the problem.

If shoes are dyed, they should be well aired for several days before being worn.

The head of a large shoe polishing and repair shop gave up the use of the poisonous dye after the case of R. G., but he had to return to the dye as the nonpoisonous dye did not give satisfactory results.

The active treatment consists of a thorough bath and rest in bed. Inhalations of oxygen may be used if the condition is extreme.

184 Alexander Street.

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AGRANULOCYTIC ANGINA *

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Several case reports during the last few years have directed attention to a peculiar blood dyscrasia characterized by a great diminution in the number of polymorphonuclear neutrophils and associated with a gangrenous ulceration of the tonsils. In 1922, Schultz [1] reported six cases and called the condition "agranulocytic angina."

The syndrome affects chiefly women of middle age and usually terminates fatally. The onset is sudden, with fever and sore throat; in several instances, it followed closely the extraction of teeth. The characteristic lesions are rapidly spreading ulcers invariably present on the tonsils and pharynx, often on the gums, buccal walls and tongue and about the genitals. There is usually little if any enlargement of the regional lymph nodes, and in about one half of the cases icterus is present. The blood shows a relative lymphocytosis with a disappearance of all or almost all of the leukocytes of the granular or myelogenous series. Smears made from the lesions in the majority of cases show Vincent's spirilla and fusiform bacilli, although a variety of organisms, such as hemolytic streptococci, staphylococci, colon bacilli and *Bacillus pyocyaneous* have been isolated.

Whether the condition is primarily a disturbance of the hematopoietic system with a secondary acute infection, or whether toxins or bacteria from the ulcerations are responsible for the depression of the myelogenous cells of the bone marrow, is not clear.

We have observed the condition in a child who on admission to the hospital presented Mikulicz's syndrome with splenomegaly and secondary anemia, and who died four months later with the classic signs of agranulocytic angina.

History.—J. J., aged 6 years, was admitted to the hospital on March 29, 1927, having been ill four days with fever, malaise and swelling of the upper eyelids and of the sides of the face.

Examination.—The eyes were half closed because of the enlarged lacrimal glands which were freely movable and painless. The parotid glands were enlarged in the upper part in front of the ears, but the whole glands did not seem to be

* Received for publication, March 3, 1928.

* From the Departments of Pediatrics and Pathology, University of Kansas School of Medicine and the Children's Mercy Hospital.

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involved. The submaxillary salivary glands, the submental lymph node and the right epitrochlear were enlarged. The cervical, axillary and inguinal lymph glands were not affected. The spleen was slightly enlarged, and a mild degree of icterus was noted. The results of further examination were essentially negative.

Laboratory Examinations.—A few granular casts were found in only one of many specimens examined. Blood counts revealed: erythrocytes, 3,400,000; total leukocytes, 5,600; hemoglobin, 70; neutrophils, 42 per cent; small lymphocytes, 50 per cent; large lymphocytes, 5 per cent, and transitionals, 3 per cent.

Cultures from the throat were negative for diphtheria bacilli. A roentgenogram of the head disclosed an infection of the right antrum.

Course.—The patient had a septic temperature curve for two weeks with frequent bleeding from the nose due to an ulcer of the septum. It was felt that the infection of the antrum was responsible for the elevation in temperature, and appropriate treatment was given. The anemia increased, the spleen became larger, and the patient was given two transfusions of blood during the first two weeks in the hospital. With the disappearance of the rise in temperature, the patient gradually improved, the swelling of the lacrimal and salivary glands diminished, and at the end of the fourth week had almost entirely disappeared. In the meantime, the blood picture changed as follows: one week after admission, erythrocytes, 2,240,000; leukocytes, 5,700; hemoglobin, 50; neutrophils, 29 per cent; lymphocytes, 65 per cent; eosinophils, 3 per cent; transitional forms, 3 per cent. At the end of the fourth week, the erythrocytes had decreased to 2,240,000, the leukocytes to 4,200 and the hemoglobin to 35, without any significant change in the differential count.

In spite of the progressive anemia, the child felt much better and was able to be up and about the wards. At the end of the fifth week, he was discharged to the outpatient department. Two weeks later, he returned for observation; the spleen had not increased in size, there were no enlarged glands, the weight was normal, the appetite was good and he was apparently doing well.

Second Admission to Hospital.—On May 23, 1927, three weeks after discharge, the patient returned to the hospital, having been ill four days with toothache, swelling of the left lower jaw and fever.

Examination.—The mouth was opened with difficulty. In the region of the lower second molar on the left side the gum was swollen, painful and bled easily. The throat was inflamed and edematous, and a gray membrane covered the left tonsil. Vincent's spirillae and fusiform bacilli were found in the smears, and the cultures were negative for diphtheria bacilli. He was given diphtheria antitoxin. Two days later, there was no improvement in the symptoms referable to the throat, and the region of the left tonsil became so swollen that it was incised. A small amount of bloody fluid escaped. The cervical, axillary, epitrochlear and inguinal glands were enlarged. The spleen was larger than at previous examinations, being a handsbreadth below the costal border, and the liver was definitely enlarged. The skin presented a yellow tint with prominence of the superficial veins. The heart and lungs were normal. The urine contained a few granular casts.

The blood count showed: erythrocytes, 2,600,000; leukocytes, 11,800; neutrophils, 12 per cent; lymphocytes, 88 per cent, many being immature. Many smudges were seen and an occasional myeloid cell.

Course.—The patient was given neoarsphenamine three times a week, and perborate of soda solution was used in the mouth. He also received a transfusion of blood weekly. Bleeding from the nose occurred frequently and copious

vomiting of blood occasionally. Both ears began to discharge a thin, serous exudate which contained Vincent's organisms. The membrane disappeared from the tonsil, leaving a necrotic area with a fetid odor. The tonsil pillars, soft palate, larynx, tongue and gums soon became involved in this gangrenous ulceration. The temperature continued between 102 and 104 F. for the next three weeks and then gradually returned to normal with elevations during the afternoon. The swelling of the throat and cervical glands disappeared after about one month, and general improvement followed. At this time, it was found that the left tonsil and part of the soft palate had sloughed away. The right tonsil did not suffer this necrosis, and apparently returned to normal.

The leukocyte count in the meantime fell from 11,800 to 2,800 and remained at this level for nearly a month, and the polymorphonuclear neutrophils disappeared from the blood.

About the end of the second month of his illness, the patient was so much improved that he was able to walk about the ward. One day during this convalescent period he complained of a recurrence of the toothache, and the dentist extracted the tooth. It was a swelling over this same tooth that brought the child to the hospital. Immediately after the extraction all the former symptoms returned. The throat became swollen and edematous, the cervical glands enlarged, and the temperature ranged from 103 to 105 F. until death, which occurred twelve days later. The last blood count made three days before death showed 1,700,000 erythrocytes and 2,000 leukocytes, all of which were mononuclear cells.

Necropsy.—Gangrenous stomatitis was present, and the regional lymph glands were enlarged. The base of the tongue, epiglottis and pharyngeal epiglottic folds had undergone extensive necrosis. Necrotic areas were found throughout the entire esophagus. The mucosa of the stomach was injected, and small ulcers were scattered over its surface. These structures gave off a foul odor. Sections through the tongue and esophagus showed large ulcerations covered with masses of bacteria and purple-staining necrotic debris. The ulcers showed an abrupt transition from normal epithelium with undermined edges extending some distance into the underlying tissue and in some places involving the mucous glands beneath. There was little leukocytic reaction about the ulcers, and only mononuclear leukocytes were found. Beneath the uninvolved epithelium there was considerable lymphocytic infiltration. Masses of bacteria invaded the submucous glands. The same character of ulcerations extended into the stomach.

Sections through a hemolymph node showed an increase of red blood cells in the sinusoids, and no polymorphonuclear neutrophils were found. In the cervical lymph glands there was considerable breaking up of the reticulum and proliferation of the endothelial cells, some of them containing pigment.

The liver weighed 1,050 Gm. and measured 17 by 16 by 10 cm. The cut surface showed indistinct lobulation. Numerous white spots, the size of a pinhead, and several larger areas of necrosis were seen. Histologically, bacteria were found in Kupffer's cells and in many of the larger vessels. Large areas showing disintegration of the cells of the liver and masses of invading bacteria were found. Bacteria filled the portal veins in many places, and there was some mononuclear leukocytic infiltration into the portal spaces.

The spleen weighed 220 Gm. and measured 14 by 8 by 6 cm. It was gray, and the capsule was thickened. The pulp was fibrotic, and the malpighian bodies were not distinct. Microscopically, there were patchy areas of hemorrhage, many monocytes and disintegration of the endothelial reticulum. Many giant cells, apparently large endothelial phagocytes and areas of necrosis of various sizes were observed. Under a lens of high magnification the latter

were found to be masses of bacteria. No polymorphonuclear leukocytic reaction was found. These areas of necrosis containing bacteria were numerous and unevenly distributed throughout the pulp. The normal markings were indistinct, and the stroma was pale-staining and rather loose.

The lungs weighed 270 Gm. They were blue-gray and crepitated and fibrinous adhesions were present on the posterior surface at both bases. The hilum glands were hyperplastic. The interlobular vessels as well as the interalveolar capillaries were full of bacteria. There was some mononuclear infiltration about the larger bronchi and desquamation of the bronchial epithelium.

The suprarenals together weighed 6 Gm., and showed hemorrhages in the medulla. Bacterial emboli were found in the small vessels of the medulla with lymphocytic infiltration about the larger vessels. The cortex was poor in lipoid, and spotted areas of degeneration were noted. In the outer layer of the cortex, masses of bacteria were found in the sinusoids.

The heart weighed 95 Gm., and the muscle was soft and flabby. Histologically, the muscle fibers presented areas of necrosis. Some of the fibers were pulled apart and showed unevenness of staining. Clumps of bacteria had infiltrated the broken muscle fibers adjacent to the small vessels. In some areas, partial necrosis of the muscle fibers was found with masses of bacteria replacing the broken strands. Only a little leukocytic reaction was present, and polymorphonuclear leukocytes were not seen.

The kidneys together weighed 260 Gm., and there was a marked subscapular congestion. The surface of both kidneys was studded with numerous small white spots. On section, the line between the cortex and medulla was found to be fairly distinct, and when cut bulged out from under the capsule. The glomeruli were visible. The ureters were normal. Microscopically, there was cloudy swelling and vacuolization of the epithelium of the tubules. The endothelium of the glomeruli was swollen and contained bacterial emboli, as did also the afferent vessels. Between the tubules some of the capillaries were plugged with bacteria. Where the tubule epithelium was undergoing degeneration, some of the epithelial cells seemed to be phagocytising the bacteria.

The bone marrow was unusually cellular, and the vast majority of the cells were mononuclear leukocytes. Some degenerating cells showing karyorrhexis were encountered. Occasional clumps of bacteria were found and also some cells of the myelocytic series.

The brain was normal, except for slight subpial injection.

Methylene blue and Gram stains were used on many of the tissue sections. The ulcerations of the mouth, pharynx and esophagus showed a variety of organisms. In other organs, especially the kidneys, liver and spleen the bacteria were all gram-positive cocci, many in chains of four or five, and all of approximately the same morphology. Cultures from the blood taken post mortem and from the bone marrow and spleen did not give any growth.

Anatomic Diagnosis.—The condition was diagnosed as acute gangrenous stomatitis, esophagitis and gastritis; cervical cellulitis; focal necrosis and bacterial embolism of the heart, liver, spleen, kidneys and suprarenal glands; acute nephritis, hepatitis and myocarditis; secondary anemia; lymphoid hyperplasia of the bone marrow and streptococci bacteremia.

COMMENT

The symptoms exhibited in this patient at the time he was first admitted to the hospital indicated that the blood-forming organs were

primarily responsible for the peculiar changes in the blood. The presence of swelling of the salivary gland with splenomegaly has been observed in patients who have later developed lymphatic leukemia. Campbell Howard,[2] in his classification of Mikulicz's disease, groups them as follows: (1) those with enlargement of the salivary glands without enlargement of the spleen or lymph glands or significant changes in the blood, (2) those with swelling of the salivary glands with splenomegaly or involvement of the lymph glands, or both, the so-called pseudoleukemia and (3) enlargement of the salivary glands with definite leukemia. Our case conforms to those of his second classification, which he regards as cases of potential leukemia. Although clinically a condition of aleukemic leukemia was considered, the postmortem observations in our patient showed that leukemia was not present.

A review of the reported cases shows a variety of changes in the spleen, marrow and lymph glands, and consequently a divergence of views regarding the cause and nature of the disease. The early writers on this subject stressed the presence of multiple septic foci at necropsy. In 1904, Schwarz reported before a medical society in Vienna an unusual case of leukopenia with a decrease in neutrophils in a boy, aged 9, with abscess of the kidney. Turck [3] referred to this case a few years later in a paper reporting similar changes in the blood in certain cases of sepsis. These patients had stomatitis, gingivitis, membranous tonsillitis and petechial hemorrhages without enlargement of the glands or spleen. Blood cultures were sterile, but the postmortem changes were those of general sepsis. In Schwarz's case of abscess of the kidney, the total leukocyte count was 600 per cubic millimeter. In one of Turck's cases, the leukocytes numbered 940 per cubic millimeter, and only one neutrophil was found in 359 leukocytes counted, or 0.28 per cent.

Aside from the case reported by Schwarz, in which a postmortem examination was not made, we have been able to find in the literature only one other report of the disease occurring in childhood. Rudolf Bantz [4] described a classic case in a boy, aged 4½ years. The patient had a marked injection of the pharynx with a membrane covering the right tonsil and a fetid odor of the breath. He was given diphtheria antitoxin. The spleen was enlarged, and a few small hemorrhagic areas were present on the back. The blood count showed 3,900,000 erythrocytes, 6,700 leukocytes, 92 per cent small lymphocytes and 8 per cent large lymphocytes. A few days later, there were 2,300 leukocytes with 93 per cent small lymphocytes and 7 per cent granulocytes which gave a negative oxidase reaction. Blood cultures were sterile. Spirillae and

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fusiform bacilli, staphylococci and streptococci were found in the throat. Examination at necropsy revealed gangrenous ulceration of the tonsil and pharyngeal walls with subpleural and subpericardial hemorrhages.

Although these early cases were not thought to be due to the infection of the throat and the so-called angina was not stressed, it is probable that they were identical with the syndrome described by Schultz nearly twenty years later as agranulocytic angina.

As Vincent's organisms are found in a great number of cases of mild infections of the throat of short duration, one hesitates to ascribe to them the power to bring about the changes in the blood and the toxemia that occur in agranulocytosis. In this connection, it is interesting to note their frequent presence in the mild infection of the throat that accompanies infectious mononucleosis. This disease, described in 1920 by Sprunt and Evans,[5] is characterized by a transient lymphocytosis, fever and enlargement of the spleen and lymph glands, terminating in recovery in three or four weeks. Longcope[6] found a decrease of the granular cells in this condition as low as 2,000 per cubic millimeter. Bloedorn and Houghton[7] found spiral organisms in three of their four cases, and they regard the disease as a form of Vincent's angina. Deussing[8] described three cases in boys beginning with "diphtheria-like" sore throat. In all of these cases there was inflammation of the upper respiratory tract; in some, there was also a membrane of the tonsils. The predominant organisms from the smears were spirillae and fusiform bacilli. The leukocytosis was due to an increase in the lymphocytes, many of them immature.

The work on laboratory animals showing a decrease in neutrophils following injections of cultures of *B. pyocyaneous* is the only direct evidence of a bacterial cause of agranulocytosis. Beatrice Lovett[9] reported the first case in America in 1924, and she isolated this organism from the ulcerations and from the tissues after death. After a sterile exudate was produced in the peritoneal cavity of the guinea-pig, the bacteria were injected. At the death of the animal, disintegration of the neutrophils was found. There was also a decrease in the number of neutrophils in the blood.

The possibility of spirillae or other organisms producing a disturbance of the blood-forming organs is suggested in Hill's[10] report on

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the development of acute leukemia following infection of the jaw after the extraction of a tooth. Two cases showed a marked increase in leukocytes; in the first there were 366,000, all mononuclears; in the second, 245,000, of which 96 per cent were mononuclears and 4 per cent neutrophils. A third patient with a similar infection developed a leukopenia with a total leukocyte count of 400, all of which were lymphocytes, a typical agranulocytic angina. It appears that in these cases identical infections of the mouth resulted in widely different hematologic reactions.

Several of the cases reported began with a fulminating angina in persons previously healthy and terminated fatally within a few days with the characteristic agranulocytosis. In these cases, it seems unlikely that there was a primary disturbance of the blood-forming organs. The average duration of the illness in the cases of agranulocytic angina collected by Kastlin [11] was from four to eight days, with extremes of from two to forty days.

Our patient lived two months after the diagnosis was made and apparently was improving, being able to leave his bed and walk about the wards, until the extraction of a tooth precipitated a return of all his symptoms. He lived twelve days after this, and it is probable that the extensive infiltration of bacterial emboli and many of the tissue changes occurred during this time.

The tissue changes are essentially those of septicemia with little reaction of the defensive forces. In the present case, bacterial emboli filled the spleen, liver, heart muscle and kidneys.

SUMMARY

1. A boy, aged 6 years, while under observation for anemia with enlargement of the salivary glands and spleen, developed Vincent's angina, leukopenia and a loss of polymorphonuclear cells in the blood.

2. Evidence of streptococcic septicemia was found at necropsy; bacterial emboli had invaded the heart muscle, spleen, liver and kidneys.

3. Before the patient died, the neutrophils completely disappeared from the blood and were not found in the tissue reactions in the inflammatory zones at necropsy.

4. There was no extensive lymphocytic or myeloid cell infiltration of the organs such as occurs in leukemia, and consequently no evidence to support the hypothesis that this disease was a primary disturbance of the myelogenous marrow tissues, and that the inflammatory condition was secondary due to lack of resistance caused by the absence of granulocytes.

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Progress in Pediatrics

SURVEY OF THE COMMUNICABLE DISEASE SITUATION IN CHICAGO*

JESSE R. GERSTLEY, M.D., Chairman

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I. S. FALK, PH.D.

I. A. ABT, M.D.

CLIFFORD G. GRULEE, M.D.

AND

JOHN F. NORTON, PH.D.

CHICAGO

OUTLINE

- Statement of Chairman
- Diseases Considered
- General Situation
  - Incidence of Communicable Disease in Chicago
  - Venereal Disease
  - Amebic Dysentery
  - Seasonal Distribution of Communicable Disease.
  - Incidence of Communicable Disease in Hospitals
  - Communicable Disease with Reference to Concentration of Population. (Accompanied by map)
  - Trend of Communicable Disease. (Special study by I. S. Falk, Ph.D.)
- Methods of Combating Communicable Disease
  - Hospitalization
    - Number of Hospitals
    - Number of Hospitals for Contagious Diseases
    - Number of Contagious Disease Beds
    - Venereal Disease Hospitals and Clinics
    - Expense at Various Hospitals
    - Cost of transportation
    - Results of Hospitalization
  - Measures by Municipality
    - Preventive Inoculations. (Report by Alton S. Pope, M.D., Chief, Bureau of Communicable Diseases of Chicago)
      - Diphtheria
      - Measles
      - Scarlet fever
      - Typhoid fever

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*Report of a special committee appointed by the Institute of Medicine of Chicago.

School Inspection. (Report by Hugh O. Jones, M.D., Assistant Commissioner of Health of Chicago)
Diphtheria in schools
Scarlet fever, measles, whooping cough
Smallpox
School vaccination
Disinfection of Schoolrooms
Milk Supply. (Report by George W. Putnam, M.S., Bureau of Dairy Products, Chicago)
Water Supply. (Report by Arthur E. Gorman, M.S., Former Chief Sanitary Engineer, Chicago
Quality of water supply
Sources of pollution of water supply
Filtration for improvement of quality
Sanitary control of swimming pools and bathing beaches
Education of Medical Students
Education of Nurses
Report from Laura R. Logan, R.N., Dean of Illinois Training School for Nurses
Report from Sister M. Lidwina, R.N., Directress of Nurses, Mercy Hospital
Report from Dora C. Saunby, R.N., Principal School of Nursing, Michael Reese Hospital
Report from M. Helena McMillan, R.N., Director School of Nurses, Presbyterian Hospital
Special Report by Charlotte Johnson, R.N., Durand Hospital of the John McCormick Institute for Infectious Diseases
Quarantine
Recommendations of Committee

STATEMENT OF CHAIRMAN

To the Institute of Medicine of Chicago:

We herewith submit the report of the Special Committee appointed by you to make a survey of the Communicable Disease Situation in Chicago. The report covers the years 1925 and 1926 and, in a measure, 1927.

As chairman of the committee I wish to acknowledge our special indebtedness to Dr. Herman N. Bundesen, former health commissioner of Chicago. Without his cooperation this great mass of statistics, much of which is now published for the first time, would not have been available. All statistics, charts and curves furnished to us by the health department are transmitted to you by his authorization.

It is only right also to express our special indebtedness to Dr. J. C. Geiger and Dr. I. S. Falk. Through their association with the Chicago Department of Health, and from their large experience, not only were they able to suggest statistical studies which proved of interest but they obtained these statistics promptly and efficiently. The completion of our report is due largely to their interested assistance.

JESSE R. GERSTLEY, M.D., Chairman.

DISEASES CONSIDERED

In the survey of the communicable disease situation the committee decided to limit itself to the following diseases:

Amebic Dysentery
Cerebrospinal meningitis
Chickenpox
Diphtheria
Encephalitis
Erysipelas
German measles
Influenza
Measles
Paratyphoid fever
Pertussis
Pneumonia
- (*a*) Combined forms of pneumonia (only in the last few years have the two forms, bronchopneumonia and lobar pneumonia, been separated)
- (*b*) Lobar pneumonia
- (*c*) Bronchopneumonia and unspecified forms of pneumonia

Poliomyelitis
Puerperal sepsis
Rabies
Scarlet fever
Smallpox
Typhoid fever

We have not considered in any detail the subject of tuberculosis. That in itself would require a special study.[1]

THE GENERAL SITUATION

INCIDENCE OF COMMUNICABLE DISEASE IN CHICAGO

During the year 1926, 7,602 deaths from contagious diseases were reported in the city of Chicago. These were distributed as indicated in table 1.

VENEREAL DISEASE

As regards venereal disease, a few statistics were obtained, but it is self-evident that the records of this disease are as yet incomplete. We submit statistics of the cases which were reported.

Venereal diseases were first made reportable in Chicago in 1918. Since that date an increasing number of cases has been reported to the health department. Table 2 gives the numbers reported from 1922 to 1926. While it is believed that a majority of clinics and physicians are faithfully reporting instances of venereal infection, it is doubtful whether such figures give a true picture of the venereal disease situation in Chicago.

1. A special report on this subject has been made by Robey, L., and Falk, I. S.: J. Prevent. M., March, 1928.

Of the cases reported in 1926, 25 per cent were among the colored population, 80 per cent among persons over 21 years old, 17.7 per cent among those from 16 to 21 years and the remainder in children under 16 years; 73 per cent were in males and 27 per cent in females; about twice as many cases were reported among single as among married persons.

AMEBIC DYSENTERY

Amebic dysentery was the subject of a recent special study, but it is interesting to note its rather frequent occurrence.

TABLE 1.—*Number of Cases and Deaths from Contagious Disease in Chicago in 1926*

Disease	Deaths	Cases Reported
Pneumonia (all forms)	3,227	9,763
Lobar pneumonia	2,050	4,323
Bronchopneumonia and unspecified forms of pneumonia	1,188	.....
Influenza	409	1,967
Diphtheria	224	2,540
Puerperal sepsis	102	62*
Erysipelas	90	839
Whooping cough	84	2,701
Scarlet fever	79	4,948
Measles	56	6,853
Cerebrospinal meningitis	29	44
Typhoid fever	24	149
Encephalitis	16	34
Amebic dysentery	11	63
Chickenpox	5	5,191
Poliomyelitis	3	28
Paratyphoid	3	11
Smallpox	2	54
German measles	0	433
Rabies	0	.....

* Incomplete reporting.

TABLE 2.—*Cases of Venereal Disease Reported in Chicago from 1922 to 1926*

Disease	1922	1923	1924	1925	1926
Syphilis	4,846	6,087	7,754	8,508	8,998
Gonorrhea	9,684	11,536	16,168	16,088	16,905
Chancroid	445	650	625	347	290
Double infections	.....	91	433	288	312
Total	14,975	18,364	24,980	25,231	26,505

SEASONAL DISTRIBUTION OF COMMUNICABLE DISEASES

Charts 1 to 11 show the seasonal distributions of certain communicable diseases. For "all causes" combined and for:

Tuberculosis	Scarlet fever	Chickenpox
Typhoid fever	Measles	Influenza
Diphtheria	Whooping cough	Pneumonia

the charts show the number of cases expected and actually reported each week of the year. (The expectancies had been computed before Jan. 1, 1926.) It will be observed that in most instances the number of reported cases were not far different from the number of expected cases.

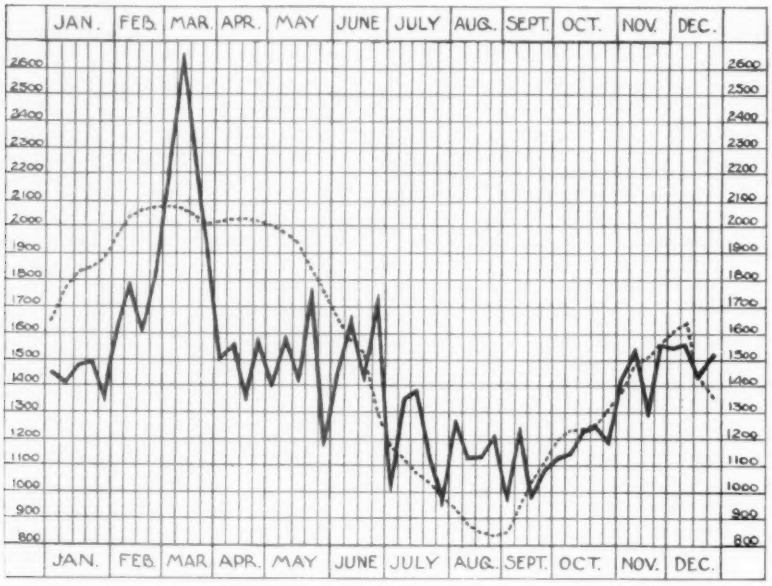


Chart 1.—Entire number of cases of communicable diseases reported and expectancy in 1926. In this and charts 2, 3, 4, 5, 6, 7, 8, 9, 10 and 11, the unbroken line represents the actual number of cases reported; the broken line represents the number of cases expected.

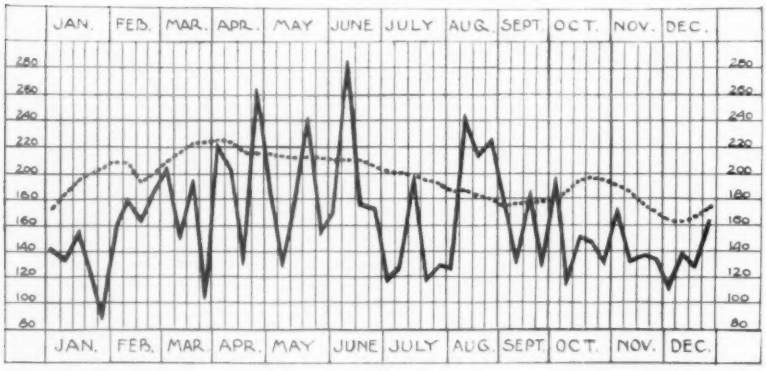
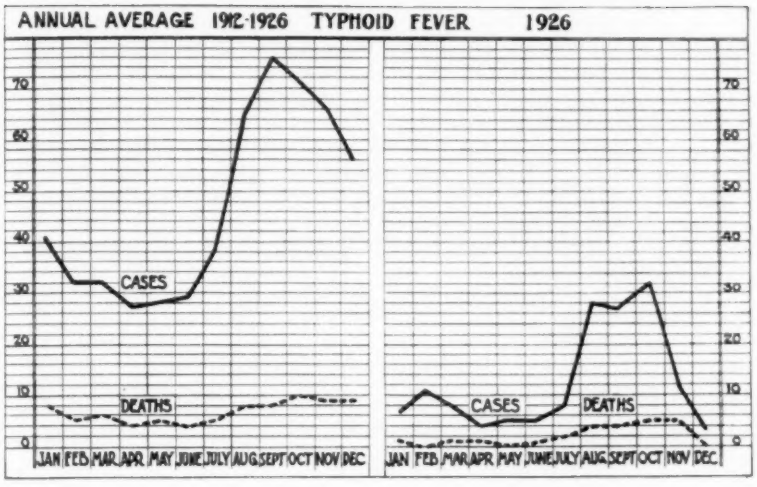


Chart 2.—Number of cases of tuberculosis (all forms) reported and expectancy in 1926.


ANNUAL AVERAGE 1912-1926 TYPHOID FEVER 1926
CASES
DEATHS
CASES
DEATHS
JAN FEB MAR APR MAY JUNE JULY AUG SEPT OCT NOV DEC
JAN FEB MAR APR MAY JUNE JULY AUG SEPT OCT NOV DEC

A B

Chart 3.—*A*, annual average of cases of and deaths from typhoid fever in the years 1912-1926; *B*, cases of and deaths from typhoid fever in 1926.

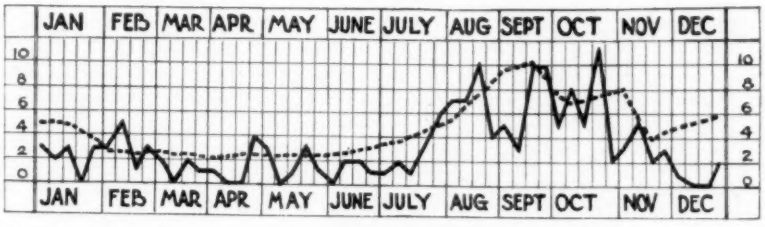


Chart 4.—Number of cases of typhoid fever reported and expectancy in 1926.

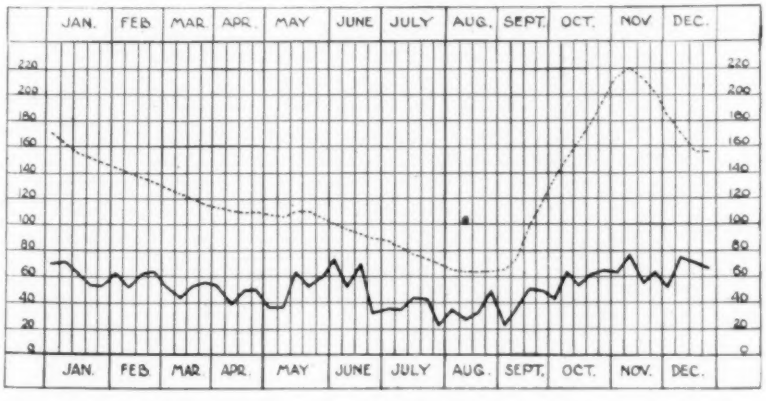


Chart 5.—Number of cases of diphtheria reported and expectancy in 1926.

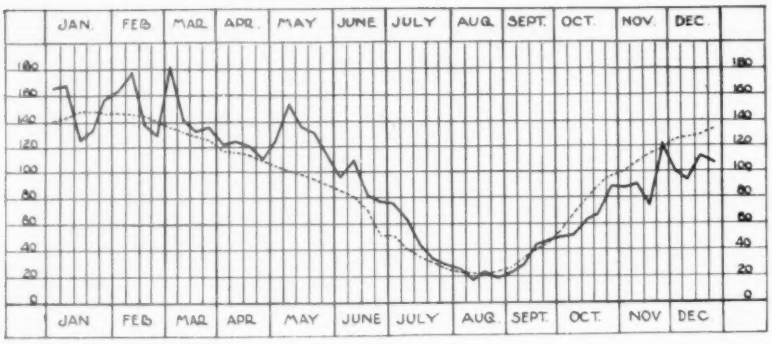


Chart 6.—Number of cases of scarlet fever reported and expectancy in 1926.

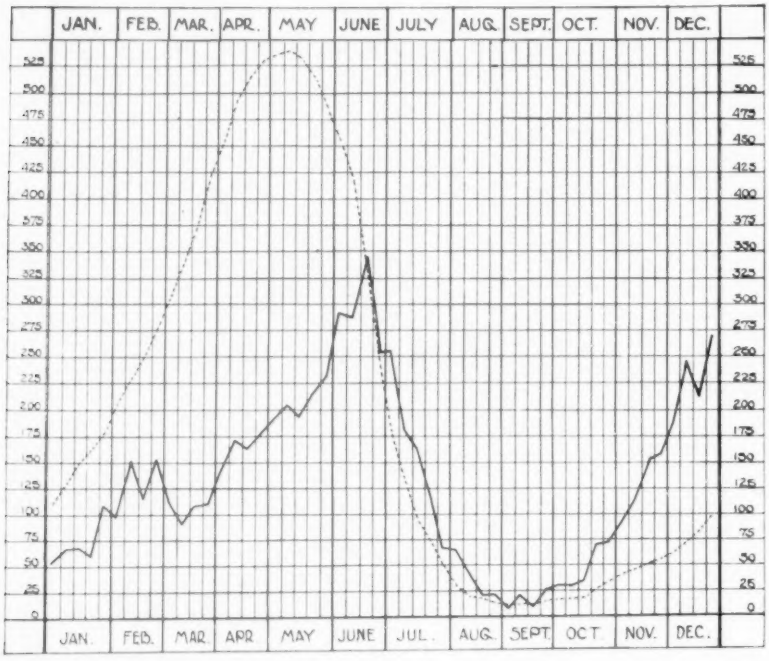


Chart 7.—Number of cases of measles reported and expectancy in 1926.

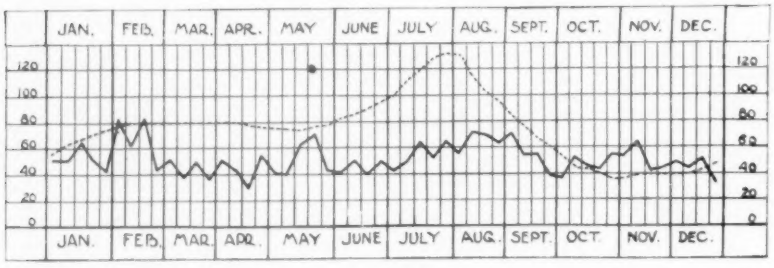


Chart 8.—Number of cases of whooping cough reported and expectancy in 1926.

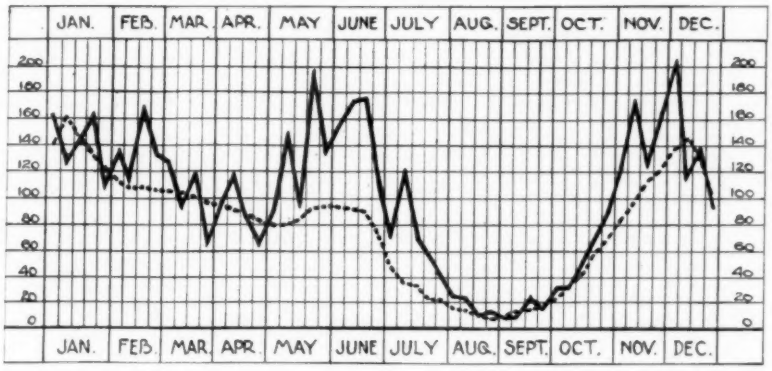


Chart 9.—Number of cases of chickenpox reported and expectancy in 1926.

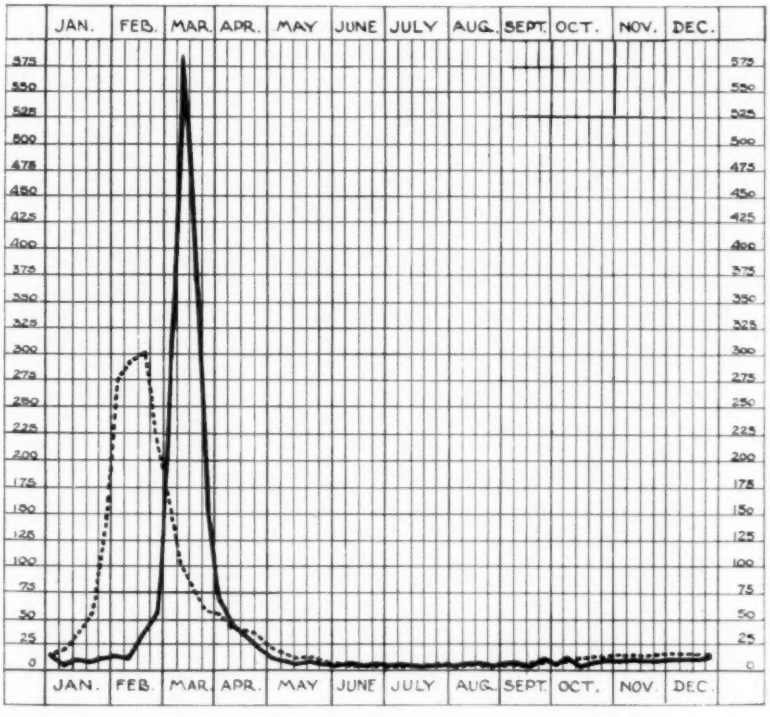


Chart 10.—Number of cases of influenza reported and expectancy in 1926.

The incidence of diphtheria was far below the expectancy and for measles and influenza the peaks came either a little earlier or a little later than had been expected.

It is significant to note from the charts how closely the incidence of communicable disease in Chicago can be predicted by statistical methods.

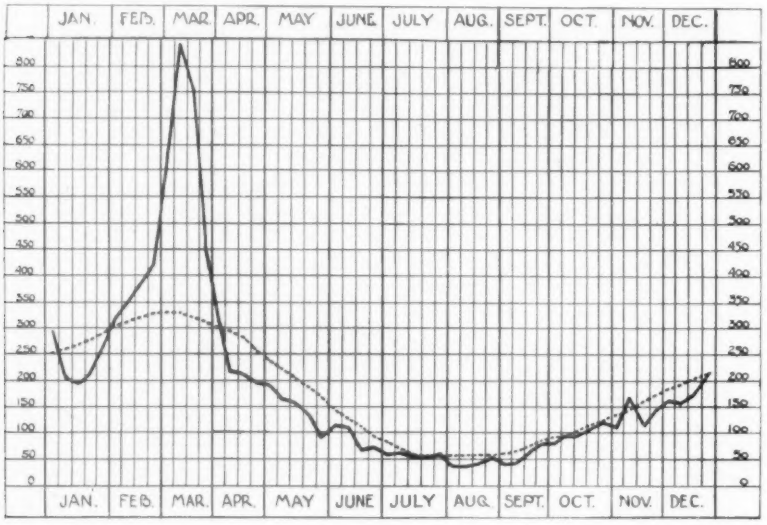


Chart 11.—Number of cases of pneumonia reported and expectancy in 1926.

TABLE 3.—*Cases of Communicable Diseases in Chicago Hospitals in 1925 and 1926*

Diseases	Cases, 1925	Cases, 1926
Chickenpox	65	177
Diphtheria	792	797
Diphtheria and whooping cough	1	0
Diphtheria and chickenpox	1	0
Encephalitis	26	4
Epidemic meningitis	5	24
Erysipelas	234	316
German measles	26	38
Measles	339	301
Poliomyelitis	31	11
Scarlet fever	2,484	2,509
Scarlet fever and diphtheria	150	33
Scarlet fever and measles	1	2
Scarlet fever and whooping cough	1	0
Scarlet fever and tuberculous meningitis	1	0
Smallpox	89	57
Typhoid fever	144	112
Tuberculosis	4,552	3,481
Whooping cough	66	85

TABLE 4.—*Report by Wards for 1926 by Bureau of Communicable Diseases, Department of Health, Chicago*

INCIDENCE OF COMMUNICABLE DISEASE IN HOSPITALS

During the years 1925 and 1926 there were 7,602 cases of communicable diseases in the hospitals with facilities for handling such cases, namely, Cook County Hospital, Durand Memorial Hospital, Municipal Contagious Disease Hospital, Isolation Hospital and the Oak Forest Tuberculosis Department. Table 3 shows the incidence, in hospitals, of each disease.

TABLE 4.—*Report by Wards for 1926 by Bureau of Communicable Diseases, Department of Health, Chicago*

	Cases Reported	Typhoid Fever	Smallpox	Measles	Scarlet Fever	Whooping Cough	Diphtheria	Streptococcic Sore Throat	Tuberculosis	Ophthalmia Neonatorum	Cerebro-spinal Fever	Infantile Paralysis	Pneumonia	Epedemic Encephalitis
Elsewhere.....	528	13	4	23	52	1	36	0	171	26	2	3	191	1
Ward 1.....	827	5	1	84	36	29	42	3	310	9	0	0	282	0
Ward 2.....	1,383	12	6	221	22	75	43	3	529	29	2	0	434	1
Ward 3.....	1,518	3	1	325	57	50	75	0	549	23	0	0	429	0
Ward 4.....	1,166	3	4	368	98	84	59	0	286	13	3	0	238	1
Ward 5.....	967	5	0	435	129	60	29	0	133	13	1	0	157	2
Ward 6.....	775	1	1	243	112	73	31	2	140	6	0	0	160	1
Ward 7.....	1,222	10	0	369	184	127	70	0	150	12	1	1	264	1
Ward 8.....	1,079	6	2	361	159	126	67	4	157	8	0	1	184	0
Ward 9.....	745	2	0	229	78	54	58	0	127	5	0	0	181	0
Ward 10.....	898	6	0	84	214	65	63	1	171	2	1	0	268	1
Ward 11.....	655	0	0	60	40	37	38	1	254	9	2	1	185	1
Ward 12.....	646	2	1	29	74	31	51	2	215	14	1	1	197	1
Ward 13.....	623	3	1	55	43	36	31	0	294	7	3	0	165	0
Ward 14.....	825	2	0	95	72	54	50	1	271	11	2	0	252	1
Ward 15.....	1,132	6	0	240	184	62	114	0	189	5	3	0	306	0
Ward 16.....	623	4	0	157	73	75	51	0	83	4	1	0	173	0
Ward 17.....	733	9	3	191	103	59	60	0	139	6	0	1	155	0
Ward 18.....	786	4	1	217	128	73	57	0	128	15	1	1	157	0
Ward 19.....	1,097	2	0	411	219	91	55	1	115	14	1	1	183	0
Ward 20.....	675	6	1	79	32	34	29	3	189	18	2	0	258	0
Ward 21.....	439	2	0	26	27	10	56	1	119	5	0	0	150	1
Ward 22.....	334	0	1	38	34	9	45	0	88	1	1	1	96	0
Ward 23.....	374	0	1	29	88	29	35	2	80	4	0	3	97	1
Ward 24.....	346	3	0	30	77	30	33	0	76	4	2	2	86	2
Ward 25.....	752	4	0	82	105	32	105	2	160	7	0	2	245	0
Ward 26.....	640	5	0	63	48	49	51	2	176	13	1	0	216	0
Ward 27.....	914	4	8	83	69	39	55	1	321	10	1	0	311	1
Ward 28.....	672	2	4	56	72	54	53	1	220	5	0	0	195	2
Ward 29.....	566	1	0	49	120	67	58	3	111	9	1	0	146	0
Ward 30.....	588	1	0	71	98	59	41	1	136	7	0	0	180	0
Ward 31.....	512	1	0	108	33	18	15	0	157	5	1	0	148	1
Ward 32.....	518	0	0	86	37	17	46	1	161	4	0	1	153	0
Ward 33.....	475	2	0	83	42	17	33	1	102	9	1	0	118	0
Ward 34.....	461	0	0	46	79	20	57	1	126	2	0	1	121	1
Ward 35.....	432	1	0	67	73	29	49	0	86	7	0	1	127	0
Ward 36.....	446	1	0	43	110	63	31	1	75	10	0	0	111	0
Ward 37.....	864	1	0	77	202	93	60	0	119	8	1	1	209	0
Ward 38.....	433	0	0	76	83	24	38	2	86	6	1	1	107	1
Ward 39.....	810	2	0	91	199	86	83	2	141	8	1	2	184	3
Ward 40.....	717	3	5	122	188	80	69	2	101	6	0	0	147	1
Ward 41.....	834	0	1	152	187	103	54	2	117	11	0	2	197	1
Ward 42.....	743	6	2	116	60	25	34	1	229	9	0	0	259	0
Ward 43.....	669	1	1	86	69	43	40	2	209	8	1	0	202	0
Ward 44.....	482	1	0	64	65	49	48	0	97	6	1	0	146	1
Ward 45.....	422	0	0	25	50	38	43	1	117	5	1	0	140	1
Ward 46.....	406	0	1	50	73	39	30	3	73	4	1	1	140	0
Ward 47.....	482	2	0	101	91	44	37	2	85	2	1	0	117	0
Ward 48.....	473	1	3	84	76	74	24	1	87	8	1	0	113	1
Ward 49.....	669	0	1	202	114	93	58	5	93	3	1	0	128	1
Ward 50.....	927	1	0	361	171	98	47	3	86	4	0	0	152	2
Hospitals.....	320	0	0	7	9	1	12	2	59	18	0	0	203	2
Total.....	**36,653**	**149**	**54**	**6,853**	**4,948**	**2,701**	**2,540**	**66**	**8,504**	**457**	**44**	**28**	**9,763**	**34**

COMMUNICABLE DISEASE WITH REFERENCE TO CONCENTRATION OF POPULATION

In tables 4 and 5 are presented certain data on the distribution by wards of certain incidences of morbidity and mortality. Considering that the populations of the several wards are approximately the same

TABLE 5.—*Deaths from Communicable Diseases During 1926, Reported by Wards*

	Typhoid Fever	Small-pox	Measles	Scarlet Fever	Diph-theria	Anterior Polio-myelitis	Cerebro-spinal Fever	Total Deaths, All Causes
Nonresidents	3	..	3	2	9	..	2	1,361
Residence not specified	2	..	3	..	4	..	..	771
Ward 1	..	..	2	1	1	..	..	767
Ward 2	3	1	2	..	5	..	1	1,185
Ward 3	..	..	3	..	8	..	1	1,243
Ward 4	..	1	1	..	9	..	1	813
Ward 5	..	..	2	5	2	..	..	706
Ward 6	..	..	..	1	..	..	..	649
Ward 7	2	..	1	5	4	..	1	772
Ward 8	1	..	2	2	4	1	..	743
Ward 9	..	..	4	..	9	..	..	631
Ward 10	1	..	2	4	2	..	1	612
Ward 11	..	..	1	..	3	..	1	586
Ward 12	1	..	..	1	4	..	1	676
Ward 13	..	..	..	4	..	..	2	558
Ward 14	2	..	..	1	3	..	..	698
Ward 15	1	..	2	2	9	..	2	962
Ward 16	1	..	2	3	6	..	..	540
Ward 17	..	..	3	1	7	1	..	596
Ward 18	1	..	3	2	5	..	2	651
Ward 19	1	..	1	2	2	..	1	772
Ward 20	1	..	2	1	3	..	..	666
Ward 21	1	..	1	1	6	..	..	571
Ward 22	..	..	1	..	9	1	..	452
Ward 23	..	..	..	3	6	..	..	488
Ward 24	..	..	..	1	3	..	..	330
Ward 25	..	..	1	..	8	..	1	668
Ward 26	..	..	2	..	4	..	1	609
Ward 27	..	..	2	1	5	..	..	979
Ward 28	..	..	..	2	2	..	..	704
Ward 29	..	..	1	..	3	..	1	672
Ward 30	..	..	1	..	2	..	..	721
Ward 31	..	..	1	1	2	..	1	560
Ward 32	..	..	1	..	9	..	..	544
Ward 33	..	..	..	3	5	..	..	563
Ward 34	..	..	..	2	3	..	..	478
Ward 35	..	..	..	2	6	..	..	465
Ward 36	..	..	1	1	4	..	..	471
Ward 37	..	..	..	2	4	..	1	777
Ward 38	..	..	1	1	2	..	..	603
Ward 39	..	..	1	2	11	..	1	857
Ward 40	..	..	..	2	5	..	..	749
Ward 41	..	..	..	2	5	..	..	823
Ward 42	1	..	2	2	..	..	1	697
Ward 43	..	..	1	1	1	..	..	673
Ward 44	..	..	..	..	2	..	..	588
Ward 45	..	..	..	3	5	..	1	648
Ward 46	1	..	..	..	1	..	1	547
Ward 47	1	..	1	4	2	..	2	609
Ward 48	..	..	..	1	2	..	1	543
Ward 49	..	..	..	2	3	..	1	679
Ward 50	..	..	1	2	2	..	..	597
Total	24	2	58	78	224	3	29	35,623

(about 60,000 persons), the number of cases in one ward may be compared roughly with the number in another ward and taken as indicative of incidence of disease. Chart 12 shows the densities of population in the several wards of the city.

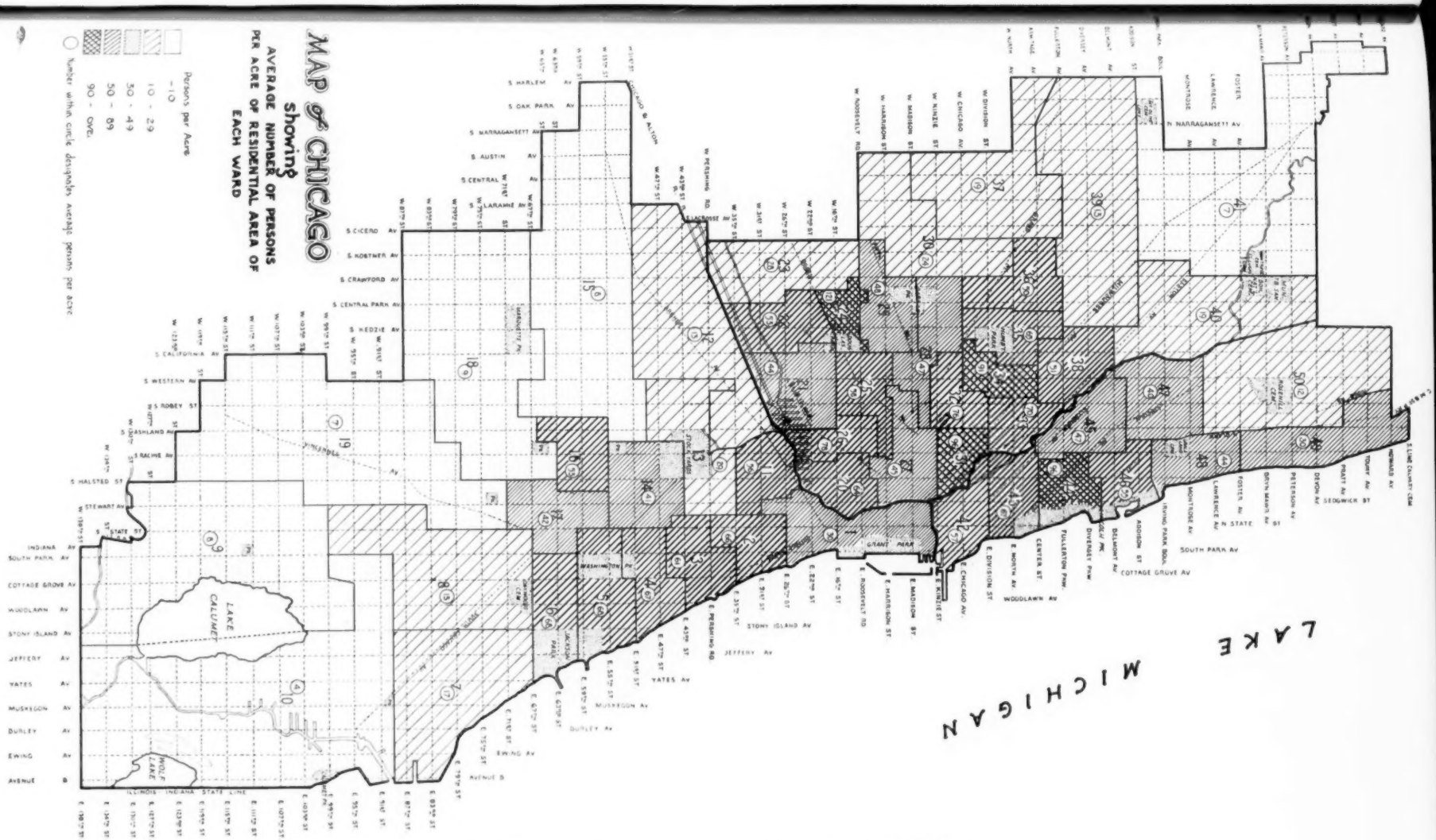


Chart 12.—Density of population in different wards of Chicago.

It will be noted from inspection of tables 4 and 5 that there are marked variations in incidence in the different wards, particularly in the figures for tuberculosis and pneumonia. These diseases are especially high in the wards with large negro populations (wards 2 and 3). Indeed, the death rates for tuberculosis and pneumonia are from three to four times as high among the negroes of Chicago as among the whites.

TREND OF COMMUNICABLE DISEASE

In a statistical study of the communicable diseases, their incidences and trends, the cases and deaths reported to the department of health have been taken for the analysis. All of the data available for the years

TABLE 6.—*Statistics Available for Certain Communicable Diseases for the Years Specified*

Disease	Cases	Deaths
1. Smallpox	1911-1926	1911-1926
2. Typhoid fever	1912-1926	1912-1926
3. Diphtheria	1911-1926	1911-1926
4. Scarlet fever	1911-1926	1911-1926
5. Measles	1911-1926	1911-1926
6. Whooping cough	1911-1926	1911-1926
7. Chickenpox	1911-1926	1911-1926
8. Poliomyelitis	1911-1926	1911-1926
9. Cerebrospinal meningitis	1911-1926	1911-1926
10. Encephalitis	1918-1926	1912-1926
11. German measles	1911-1926	.....
12. Influenza	1918-1926	1912-1926
13. Pneumonia:		
(a) Combined forms	1911-1926	1911-1926
(b) Lobarpneumonia	1920-1926	1912-1926
(c) Bronchopneumonia and unspecified forms	.....	1912-1926
14. Amebic dysentery	1926	1921-1926
15. Paratyphoid fever	1921-1926	1921-1926
16. Puerperal sepsis	1920-1926	1912-1926
17. Rabies	.....	1912-1926
18. Erysipelas	1911-1926	1912-1926

1911-1926, inclusive, have been utilized. The summary in table 6 shows the diseases considered and the periods for which data are available in a form suitable for the purposes of this study.

The original data for this study are presented in table 7.

For a ready analysis of trends the data have been arranged in graphic form. Most of the graphs show the curves of morbidity and mortality from 1911 to 1926. When the data were available for shorter periods only, these were plotted. In addition to the curves of the reported cases and deaths the charts show straight-line trends. The positions of these trend lines were calculated by the method of least squares.[2]

2. The principle of this method is that the sum of the squares of deviations above the line is approximately equal to the sum of the squares of deviations below the line (i.e., the difference of these sums is a minimum).

TABLE 7.—*Reported Cases and Deaths from Certain Communicable Diseases in Chicago from 1911 to 1926*

	Smallpox		Typhoid Fever		Diphtheria		Scarlet Fever		Measles		Whooping Cough		Chickenpox		Poliomyelitis		Cerebrospinal Meningitis		Encephalitis	
Years	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
1911	105	0	.....	...	7,669	874	7,422	471	6,520	140	951	52	2,456	0	29	11	103	40	...	..
1912	68	7	1,051	175	7,288	950	8,703	602	6,784	119	2,462	140	2,027	0	135	18	49	39	...	49
1913	136	1	1,555	249	8,593	952	10,600	906	15,132	292	1,480	101	2,860	2	24	3	51	61	...	34
1914	70	1	1,181	167	7,071	768	3,518	228	4,391	83	5,317	230	3,835	0	53	10	99	47	...	28
1915	33	0	915	132	5,863	680	3,336	77	18,964	236	1,334	58	4,831	0	31	9	91	51	...	39
1916	49	0	1,034	128	6,980	786	8,681	162	7,591	131	2,853	114	4,868	2	285	49	110	46	...	67
1917	292	2	345	43	10,290	1,216	13,444	624	19,020	245	5,156	218	5,192	0	551	187	335	198	...	41
1918	266	4	265	38	5,708	723	1,809	46	2,663	62	4,345	185	3,018	3	96	27	224	101	...	28
1919	98	0	228	31	6,361	592	4,550	116	16,075	200	3,404	141	4,091	6	110	16	108	47	60	37
1920	154	1	232	30	7,761	626	8,557	180	9,025	87	4,154	151	3,984	7	78	13	79	37	118	93
1921	246	4	252	30	9,313	683	5,450	189	8,731	120	4,118	151	4,583	2	135	30	79	31	166	49
1922	95	15	181	31	7,367	558	3,898	110	11,673	152	3,659	98	3,957	8	48	16	61	29	58	15
1923	67	1	473	53	5,836	302	3,529	82	15,168	209	3,426	136	4,604	7	86	5	55	31	70	18
1924	236	1	362	48	3,672	218	4,953	74	6,000	73	3,748	110	5,451	3	67	2	33	22	48	9
1925	70	12	236	44	2,926	240	7,668	128	13,175	116	4,496	146	3,484	12	66	7	43	28	49	19
1926	54	2	149	24	2,540	224	4,948	79	6,853	56	2,701	84	5,191	5	28	3	44	29	34	16

	German Measles		Influenza		Lobar Pneumonia		Broncho-pneumonia and Unspecified		Combined Forms of Pneumonia		Amebic Dysentery		Paratyphoid		Puerperal Sepsis		Rabies		Erysipelas	
Years	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
1911	167	...	.....	...	...	.....	...	.....	1,946	4,929	..	..	..	..	...	...	..	..	633	...
1912	136	..	.....	72	...	1,427	...	1,854	1,512	4,892	..	..	..	..	...	124	..	8	.....	128
1913	377	...	.....	85	...	1,702	...	2,048	2,574	4,871	..	..	..	..	...	95	..	2	590	118
1914	341	...	.....	117	...	1,963	...	2,054	6,422	4,077	..	..	..	..	...	119	..	5	786	102
1915	302	...	.....	287	...	2,039	...	1,785	7,706	3,569	..	..	..	..	...	72	..	1	942	119
1916	1,241	...	.....	404	...	2,293	...	1,520	9,031	3,863	..	..	..	..	...	99	..	..	1,396	171
1917	2,191	...	.....	202	...	2,995	...	2,002	9,303	5,018	..	..	..	..	...	69	..	2	1,132	140
1918	1,455	...	43,533	6,971	...	4,578	...	2,385	23,309	7,000	..	..	..	..	...	75	..	..	555	67
1919	218	...	9,237	1,757	...	1,609	...	1,674	10,820	3,353	..	..	..	..	...	54	..	1	494	60
1920	174	...	30,480	2,037	4,505	2,123	...	1,688	15,534	3,815	..	..	..	..	105	76	..	1	895	88
1921	284	...	699	114	2,548	1,087	...	1,089	8,236	2,216	..	4	12	3	108	68	..	..	775	65
1922	2,260	...	3,032	317	3,245	1,508	...	1,217	9,830	2,779	..	2	3	..	51	56	..	..	775	65
1923	234	...	2,884	428	4,479	2,075	...	1,599	11,628	3,745	..	3	3	8	55	91	..	..	992	101
1924	234	...	605	184	.....	1,747	...	1,121	9,530	2,892	..	6	6	10	107	131	..	..	819	49
1925	2,162	...	1,100	270	4,308	1,917	...	1,120	9,467	3,075	..	4	4	8	84	80	..	..	765	85
1926	433	...	1,967	409	4,323	2,050	...	1,188	9,763	3,227	63	11	11	3	62	102	..	..	839	90

TABLE 8.—*Reported Cases of Certain Communicable Diseases in Chicago in 1926 and the "Expected" Cases for 1927*

No.	Diseases	Cases Reported in 1926	Trend	Cases Expected in 1927 by Least Square Method	Cases Expected in 1927 by 9 Year Median Weekly Basis Normal Expectancy
1	Pneumonia (all forms)	9,763	Down	8,100 *	9,513 ††
2	Measles	6,853	Up	12,000 † ‡	16,573 †† ‡‡
3	Chickenpox	5,191	Up	4,700 *	4,063 ††
4	Scarlet fever	4,948	Down	4,500 †	4,573 ††
5	Lobar pneumonia	4,323	.....	.....	.....
6	Whooping cough	2,701	Down	3,150 *	3,625 ††
7	Diphtheria	2,540	Down	2,500 *	5,720 ††
8	Influenza	1,967	Down	1,500 §	1,466 §§
9	Erysipelas	839	Down	820 ‖	.....
10	German measles	433	Up	.....	.....
11	Typhoid fever	149	Down	210 *	228 ††
12	Amebic dysentery	63	.....	.....	.....
13	Puerperal sepsis	62		Incomplete reporting	
14	Smallpox	54	Down	90 ¶	95 §§
15	Cerebrospinal fever	44	Down	70 †	65 ††
16	Encephalitis	34	Down	30 #	.....
17	Poliomyelitis	28	Down	55 **	71 ¶
18	Paratyphoid	11	.....	.....	.....

* 10 year data (1917–1926).
† 15 year data (1911–1925).
‡ This expectancy is too low.
§ 6 year data (1921–1925).
‖ 14 year data (1913–1926).
¶ 11 year data (1916–1926).
# 7 year data (1919–1925).
** 8 year data (1918–1925).
†† 9 year data (1918–1926).
‡‡ Epidemic expectancy.
§§ 7 year data (1920–1926).

TABLE 9.—*Reported Deaths from Certain Communicable Diseases in Chicago in 1926 and the "Expected" Deaths for 1927*

No.	Diseases	Deaths Reported in 1926	Trend	Deaths Expected in 1927 by Least Square Method	Deaths Expected in 1927 by 9 Year Median Weekly Basis Normal Expectancy
1	Pneumonia (all forms)	3,227	Down	3,000 *	2,912 †
2	Lobar pneumonia	2,050	.....	.....	.....
3	Bronchopneumonia and unspecified forms of pneumonia	1,188	Down	1,000 *	.....
4	Influenza	409	Down	.....	283 †
5	Diphtheria	224	Down	250 ‡	447 †
6	Puerperal sepsis	102	Down	81 *	.....
7	Erysipelas	90	Down	60 *	.....
8	Whooping cough	84	Down	80 ‡	115 †
9	Scarlet fever	79	Down	100 †	91 †
10	Measles	56	Down	120 *	100 †
11	Cerebrospinal fever	29	Down	30 *	27 †
12	Typhoid fever	24	Down	20 §	60 †
13	Encephalitis	16	Up	17 ‖	.....
14	Amebic dysentery	11	.....	.....	.....
15	Chickenpox	5	Up	7 *	.....
16	Poliomyelitis	3	Down	0	.....
17	Paratyphoid	3	.....	.....	.....
18	Smallpox	2	Up	7 *	.....
19	German measles	0	.....	.....	.....
20	Rabies	0	.....	.....	.....

* 15 year data (1912–1926).
† 9 year data (1918–1926).
‡ 10 year data (1917–1926).
§ 11 year data (1916–1926).
‖ 5 year data (1922–1926).

From an inspection of the tables and charts we have prepared the summaries in tables 8 and 9. The diseases are arranged in order of decreasing importance according to the number of (*a*) cases (table 8) and (*b*) deaths (table 9) in the year 1926. Thus, in that year (1926) there were 9,763 cases of pneumonia (all forms). Hence, this disease was listed first in table 8. Measles was second with 6,853 cases; chickenpox, third with 5,191 cases, etc. For each disease the table shows the number of cases reported in 1926, the slope of the trend line—whether "up" or "down"—and the number of cases "expected" for the year 1927, from an extrapolation of the trend line.[3] In table 9 an entirely similar analysis of deaths is presented.[4] It will be noted at once that the sequence of the causes of sickness is not the same as the sequence of

TABLE 10.—*Deaths Per Hundred Cases in Chicago in 1926—Diseases Arranged According to Decreasing Values of Fatality Rates*

Number	Disease	Cases	Deaths	Reported Deaths per 100 Reported Cases
1	Cerebrospinal meningitis	44	29	66
2	Encephalitis	34	16	47
3	Lobar pneumonia	4,323	2,050	47
4	All forms of pneumonia	9,763	3,227	33
5	Paratyphoid	11	3	27
6	Influenza	1,967	409	21
7	Amebic dysentery	63	11	17
8	Typhoid fever	149	24	16
9	Erysipelas	839	90	11
10	Poliomyelitis	28	3	11
11	Diphtheria	2,540	224	9
12	Smallpox	54	2	4
13	Whooping cough	2,701	84	3
14	Scarlet fever	4,948	79	2
15	Measles	6,853	56	1
16	Chickenpox	5,191	5	1
17	Puerperal sepsis	62	102	Incomplete reporting
18	German measles	433	0	..
19	Rabies	..	..	..
20	Bronchopneumonia and unspecified forms of pneumonia	..	1,188	..

causes of death. Pneumonia (all forms) leads both in cases and in deaths; but measles, which is the second leading cause in the table of cases, is tenth in the table of deaths; chickenpox is third in importance by the number of cases and fifteenth by the number of deaths.

It is obvious that in the evaluation of the importance of and the attention which should be given to a communicable disease, in addition to the

3. By projecting the straight lines beyond 1926, we obtain the "expected" values (cases or death) for 1927 or subsequent years. In a few cases (i.e., poliomyelitis) in which the slopes of the trend lines are markedly affected by unusual epidemic occurrences, trend lines have also been calculated for the years other than those in which the epidemics occurred.

4. The numbers of "expected" cases have also been independently computed as the median values for the data (number of cases per annum) of the past nine years.

incidence of cases and of deaths, other factors must be considered. Table 10 has been arranged to emphasize the importance of the lethality of a disease. For the purposes of this analysis, using the data of 1926, the ratio of reported deaths to reported cases has been computed and expressed as a fatality rate (deaths per hundred cases).[5] The diseases have been listed in a sequence determined by these fatality rates. Thus, cerebrospinal meningitis heads the list with a fatality of eighty-six deaths per hundred cases; encephalitis and lobar pneumonia come second with fatality rates of forty-seven. At the other extreme is found chickenpox with one death per thousand cases and measles with one per hundred.

By referring to tables 8 and 9, it will be found that:

1. For the eleven diseases for which the "expected" number of cases has been computed by two methods (table 7), there is substantial agreement in the estimates. Using the "least square" method, a total of 36,875 cases is "expected" for 1927; by the "nine-year median" method, a total of 45,992 cases for these eleven diseases. The discrepancy in the total is largely due to differences in the estimates for measles and diphtheria. It may be estimated finally that there will be not far from 40,000 cases of communicable diseases reported in the year 1927 for the causes listed.

2. Similarly, for the diseases listed in table 9, it is estimated that there will be between 4,000 and 4,200 deaths in Chicago in 1927.

3. The numbers of reported cases are, on the whole, showing declining trends for all of the diseases except measles, chickenpox and German measles.

4. The numbers of reported deaths for the several communicable diseases all show downward trends, excepting encephalitis, chickenpox and smallpox.

In the following pages each of the communicable diseases which has been studied statistically is considered specifically.

1. *Pneumonia.*—It will be observed by reference to chart 13 that:

(*a*) Pneumonia (all forms), from 1912 to 1926, has been showing a rising trend in number of cases. If only the data from 1917 to 1926 are considered, the trend is decreasing.

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5. It is almost gratuitous to remark on the cautions with which these figures must be used. For most diseases there are no reliable objective standards by which the accuracy or completeness of reporting of either cases or deaths may be judged; nor is it feasible always to judge whether the same or different degrees of accuracy or completeness apply to the morbidity or mortality from a particular communicable disease.

(*b*) The trends for the number of cases of lobar pneumonia, or for deaths from pneumonia (all forms), lobar pneumonia, bronchopneumonia and pneumonia (unspecified), and of acute and chronic bronchitis combined with pneumonia (all forms), are all declining. The last (all deaths from bronchitis and those from pneumonia combined) probably gives a correct picture of the trend of mortality from this group of respiratory diseases.

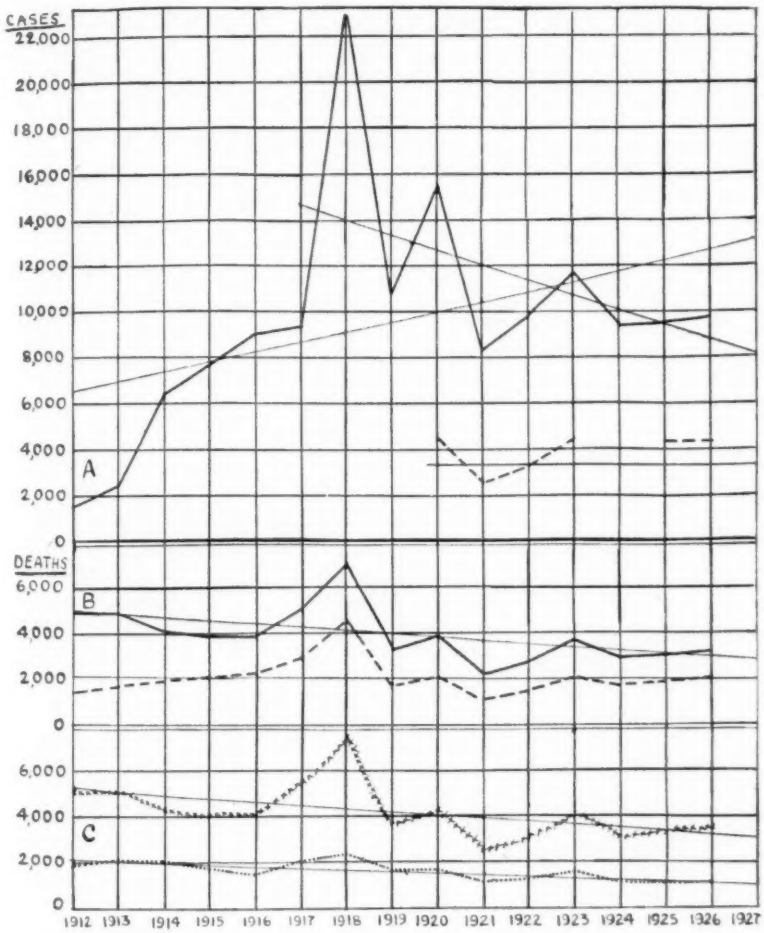

Chart 13.—Data collected on pneumonia in Chicago. In this and the following similar charts, the straight lines show the trend. *A*, number of cases; unbroken line represents all forms of pneumonia; broken line, lobar pneumonia. *B*, number of deaths; unbroken line represents all forms of pneumonia; broken line, lobar pneumonia. *C*, number of deaths, dotted line represents those from bronchopneumonia and unspecified forms of pneumonia; *x* line represents those from acute and chronic bronchitis and all forms of pneumonia.

(*c*) Approximately 10,000 cases and 3,000 deaths per annum may be expected from this group of communicable diseases.[6]

2. *Measles.*—In Chicago, measles is alternately of high and low incidence in a fairly regular two-year cycle. In the years from 1911 to 1926

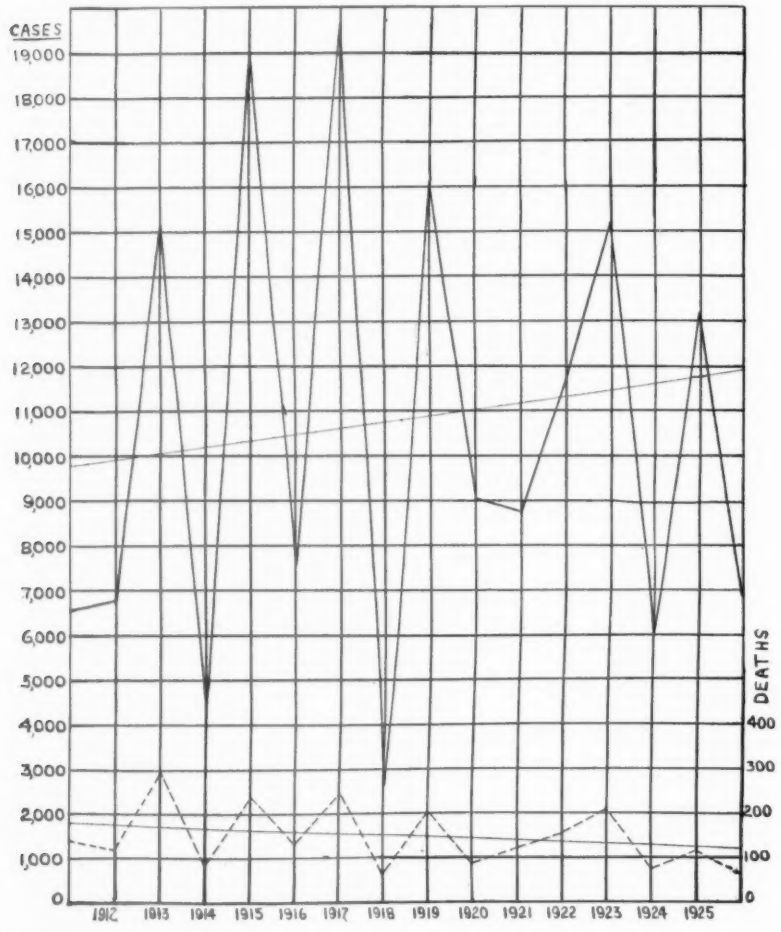

Chart 14.—Data collected on measles in Chicago; unbroken line represents number of cases; broken line, number of deaths.

the trend of the number of reported cases has been upward, and in the more recent years, 1918 or 1919 to 1926, slightly downward. The trend

6. Although there is considerable uncertainty concerning the validity of the procedure, pneumonia is treated here as a "communicable disease," because of a certain portion of the bacteriologic and epidemiologic evidences, because it is reportable as a communicable disease by the Illinois sanitary code and because it is commonly treated as such in medical practice.

of the number of deaths from measles is downward (chart 14). In a year of low prevalence, such as 1926, some 6,000 to 7,000 cases may be expected. In a year of a high incidence, such as 1927, as many as from 12,000 to 17,000 cases may be the normal number. The deaths may range from about fifty in a year of low incidence to 120 in a year of high incidence. The cases and the deaths come almost entirely among children under the age of 15 years (chart 15). The deaths are almost

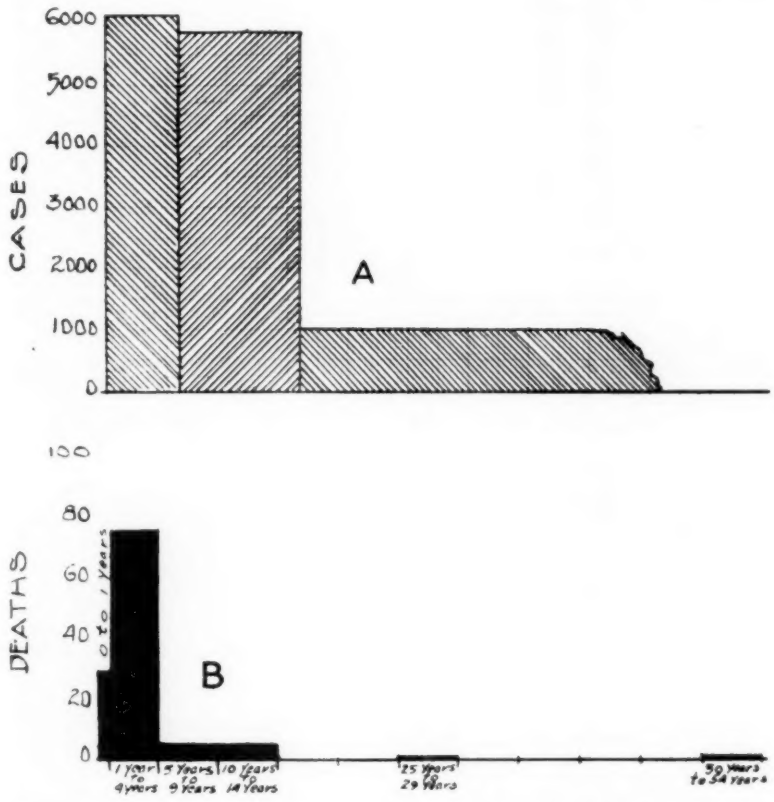

Chart 15.—*A*, cases of measles in Chicago in 1925; *B*, deaths from measles in Chicago in 1925.

entirely among children under the age of 5 years. The special danger to young children from measles is indicated by chart 16.

3. *Chickenpox.*—The numbers of both reported cases of and deaths from chickenpox show rising trends. Whether this means increasing morbidity and mortality from this disease or merely increasing completeness in reporting is uncertain. In the immediate future some 4,000 to 5,000 cases and only about five to ten deaths per annum may be considered normal (chart 17).

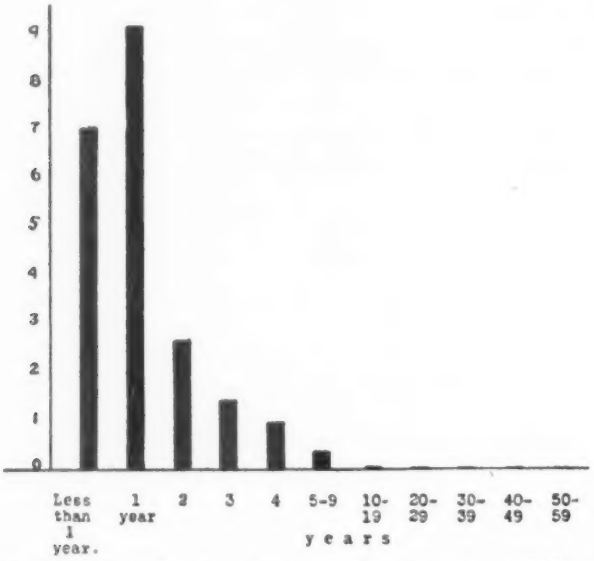

Chart 16.—Number who die from measles in every 10,000 children of the ages given.

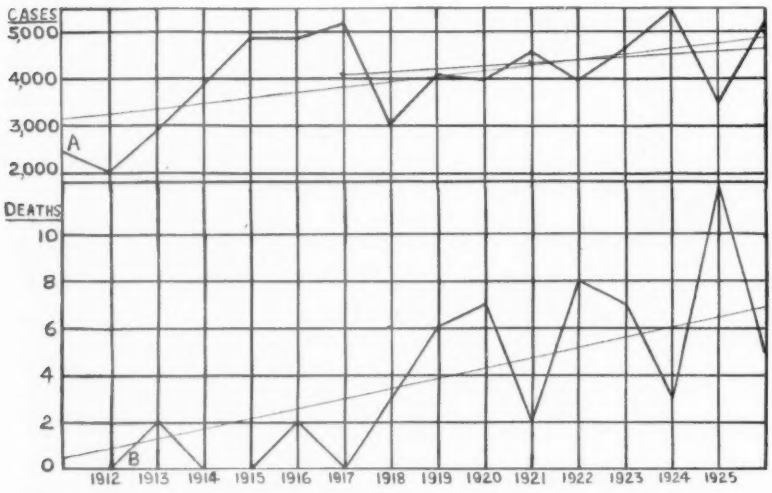

Chart 17.—Data collected on chickenpox in Chicago; *A* indicates number of cases; *B*, number of deaths.

4. *Scarlet Fever.*—The numbers of both reported cases and deaths show steeply declining trends. Forty-five hundred cases and 100 deaths per annum are estimated for 1927 (chart 18).

5. *Whooping Cough.*—For the period from 1911 to 1926, the numbers of both cases and deaths show rising trends. For more recent years (1917 to 1926), both trends are declining. The cases are estimated at from 3,000 to 3,700, and the deaths at from eighty to 120 per annum (chart 19).

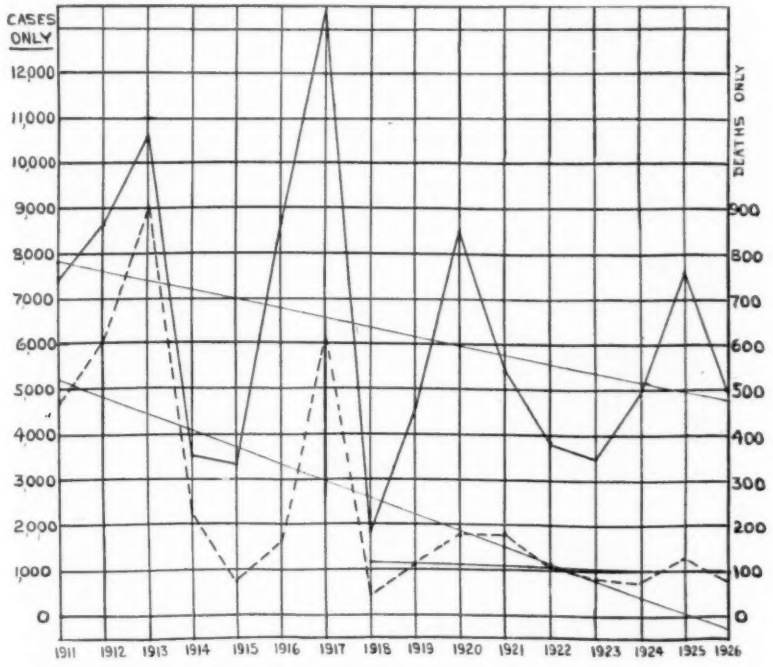

Chart 18.—Data collected on scarlet fever in Chicago; unbroken line represents number of cases; broken line, number of deaths.

6. *Diphtheria.*—The numbers of both cases and deaths show remarkably steep declining trends. Sharp breaks in the curves of incidences appeared in 1921, and nearly steady declines have continued since. Approximately 2,500 cases and from 100 to 250 deaths per annum are estimated for 1927 (chart 20).

7. *Influenza.*—It is felt that a reliable statistical evaluation of influenza incidence is not feasible. The meaning of reported interepidemic influenza is uncertain. Whatever that condition is that is reported as nonepidemic influenza, it is estimated that there will be some 1,500 cases and some 300 deaths (chart 21).

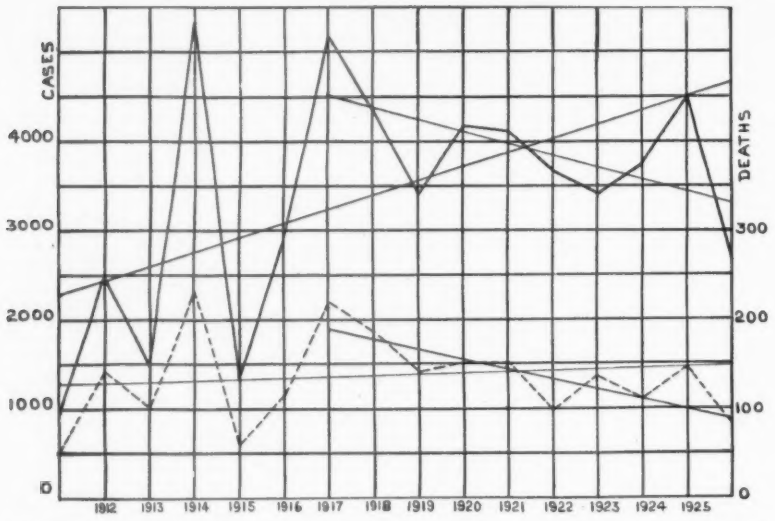

Chart 19.—Data collected on whooping cough in Chicago; unbroken line represents number of cases; broken line, number of deaths.

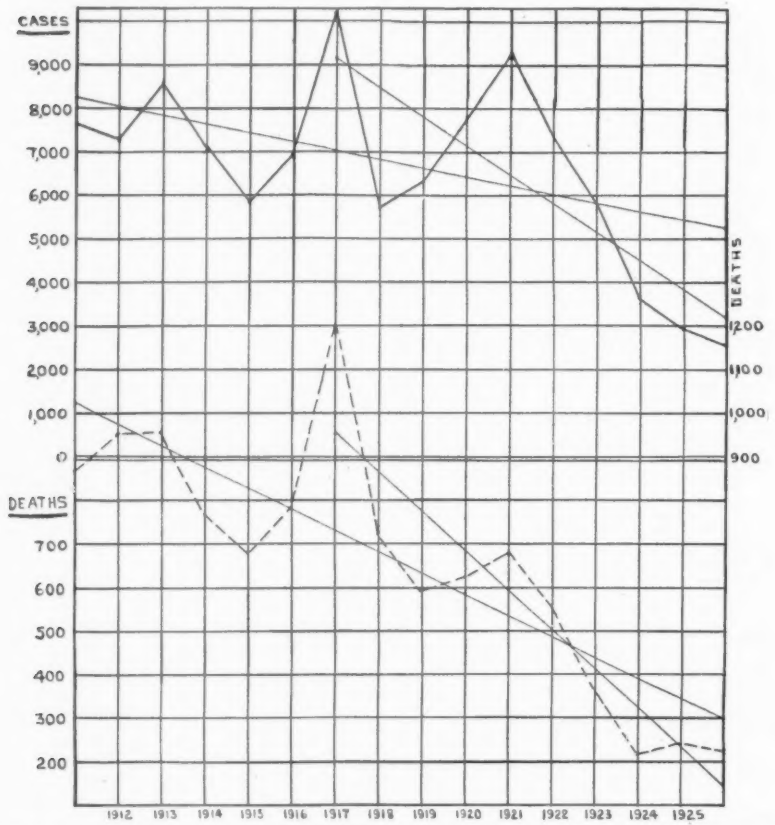

Chart 20.—Data collected on diphtheria in Chicago; unbroken line represents number of cases; broken line, number of deaths.

8. *Erysipelas.*—The numbers of reported cases and deaths show slightly declining trends. Some 800 cases and sixty deaths per annum are estimated. The recent investigations on the specific etiology of this disease may, by focusing attention on it, bring an increase in the number of cases reported in the near future (chart 22).

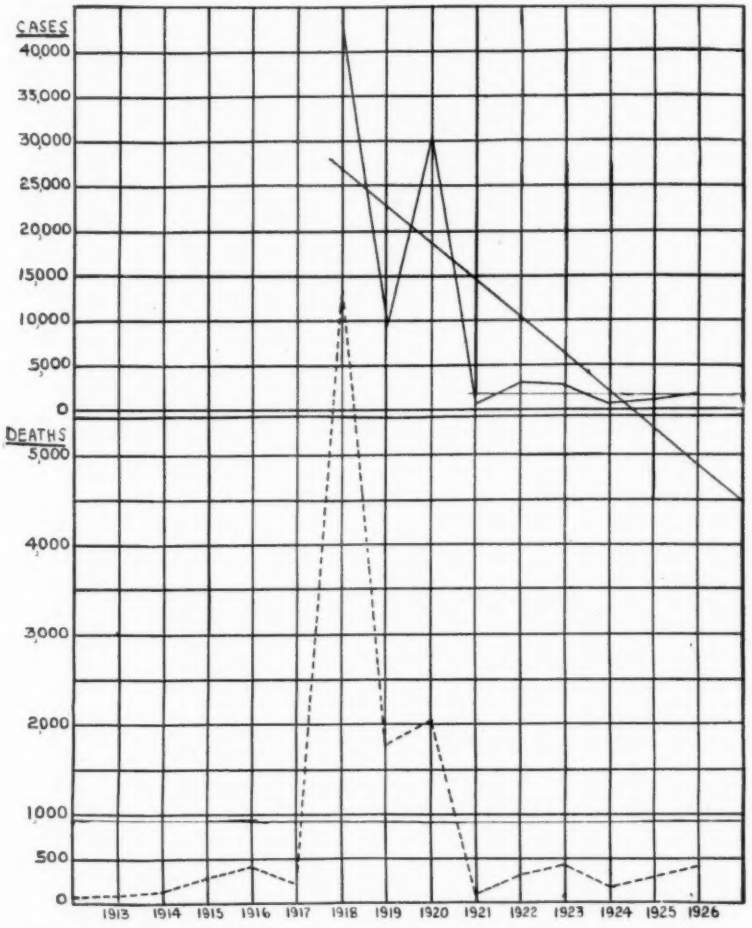

Chart 21.—Data collected on influenza in Chicago; unbroken line represents number of cases; broken line, number of deaths.

9. *German Measles.*—The number of cases reported shows an increasing trend for the period from 1911 to 1926, but an approximately horizontal trend line for more recent years. No deaths from this disease are reported. The disease has shown such a marked cyclic characteristic in its incidence, that it is extremely difficult to estimate expected incidence within the range of from 200 to 2,200 cases per annum (chart 23).

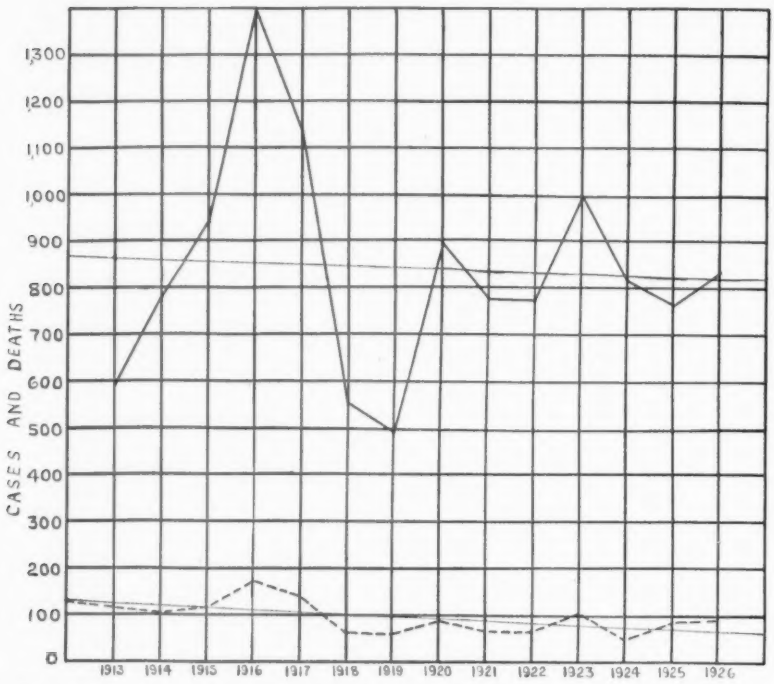

Chart 22.—Data collected on erysipelas in Chicago; unbroken line represents number of cases; broken line, number of deaths.

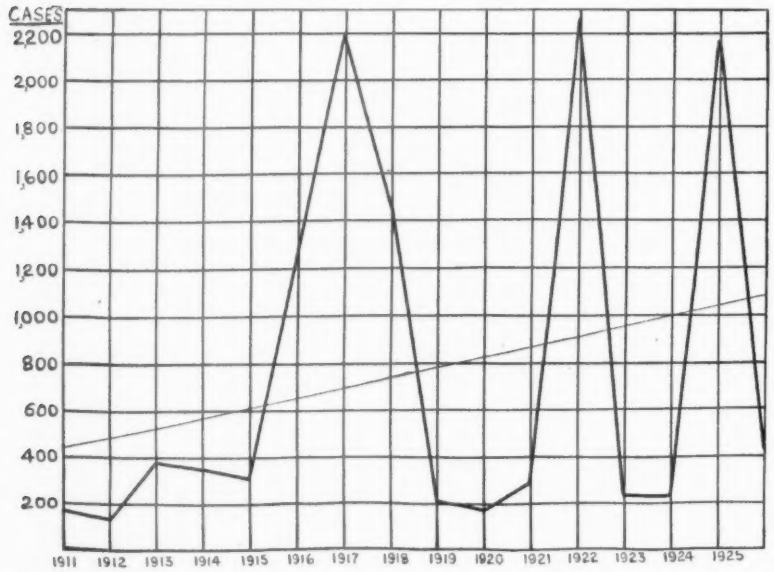

Chart 23.—Data collected on German measles in Chicago; unbroken line represents number of cases.

10. *Typhoid Fever.*—This disease has been almost entirely eliminated from Chicago's bills of morbidity and mortality. The normal expectancy in recent years has been about 200 cases and about twenty-five deaths (chart 24).

11. *Amebic Dysentery.*—Except for the data for special investigations and for rare epidemic outbreaks, adequate statistical information on incidence is not available. In 1926, sixty-three cases and eleven deaths from amebic dysentery were reported.

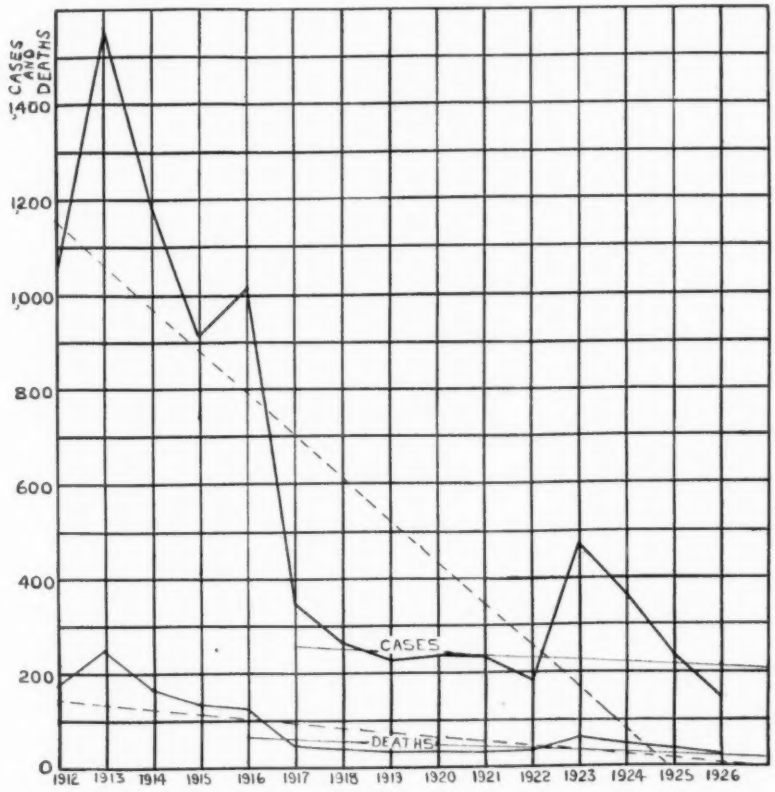

Chart 24.—Data collected on typhoid fever in Chicago; upper unbroken line represents number of cases; upper broken line, trend of cases; lower unbroken line, number of deaths; lower broken line, trend of deaths. The unbroken straight lines represent the trends computed for the more recent years only.

12. *Puerperal Sepsis.*—It is well known that physicians are seriously negligent in reporting cases of this disease. In 1926, sixty-two cases and 102 deaths were reported. The rising curve of mortality of recent years is more probably indicative of better reporting than of an increasing occurrence of deaths from this cause. The control of puerperal mor-

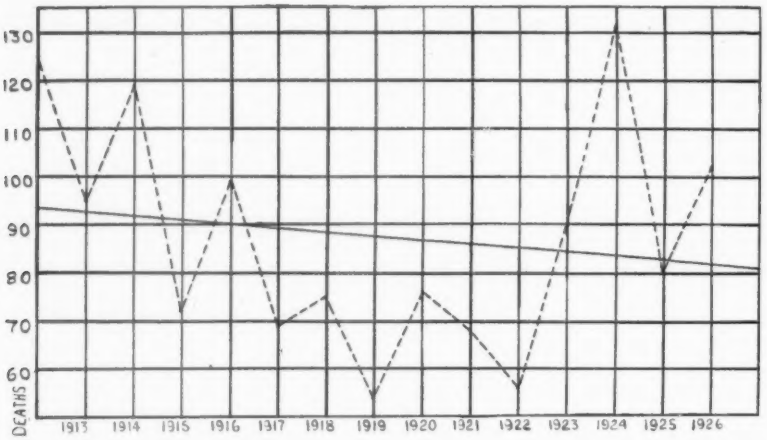

Chart 25.—Data collected on puerperal sepsis in Chicago; data on number of cases incomplete; broken line represents number of deaths.

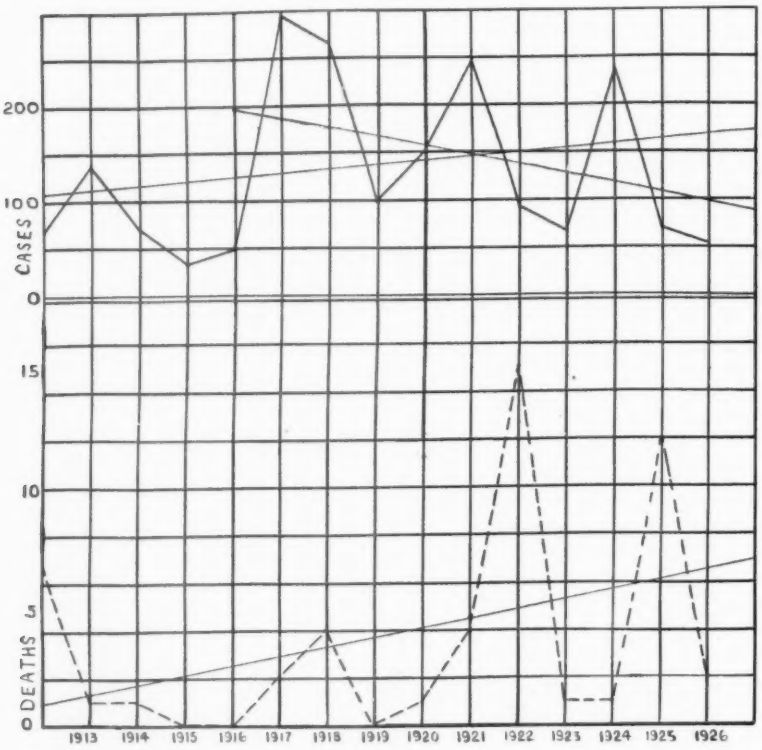

Chart 26.—Data collected on smallpox in Chicago; unbroken line represents number of cases; broken line, number of deaths.

tality is now the object of a careful survey by an advisory board to the commissioner of health (chart 25).

13. *Smallpox.*—The vital statistics of this disease for most recent years do not show significant changes in the morbidity (from seventy-five to 250 cases per annum), but a generally rising trend of mortality (chart 26).

14. *Cerebrospinal Meningitis.*—The rates of both reported morbidity and mortality have been low and declining (chart 27). The unusually

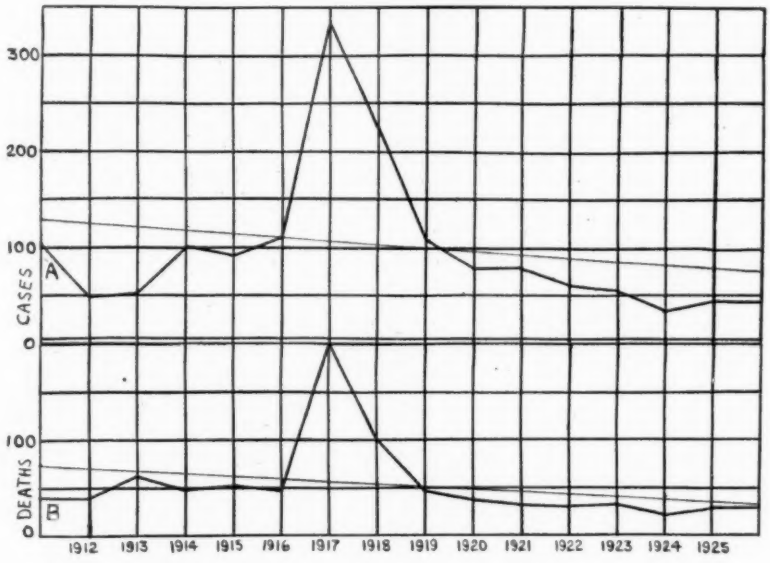

Chart 27.—Data collected on meningitis in Chicago; *A* indicates number of cases; *B*, number of deaths.

high incidence of this disease at the time of this writing (June, 1927) indicates the continued threat which this disease holds for large cities.

15. *Encephalitis.*—This disease obtained especial notice shortly after the influenza pandemic. Its reported morbidity and mortality rates have been declining since 1920 and 1921 (chart 28).

16. *Poliomyelitis.*—The reported morbidity and mortality rates have been declining steadily since 1916, the last year of unusually high prevalence (chart 29).

As these discussions are without reference to specific death rates for the several diseases, and the neglect of absolute as opposed to relative values, they must be considered with the necessary caution.

METHODS OF COMBATING COMMUNICABLE DISEASE

HOSPITALIZATION

Number of Hospitals.—There are 107 licensed hospitals in Chicago. These are:

U. S. Public Health: Marine Hospital—1.

State of Illinois: University of Illinois Hospital, Illinois Eye and Ear Hospital—2.

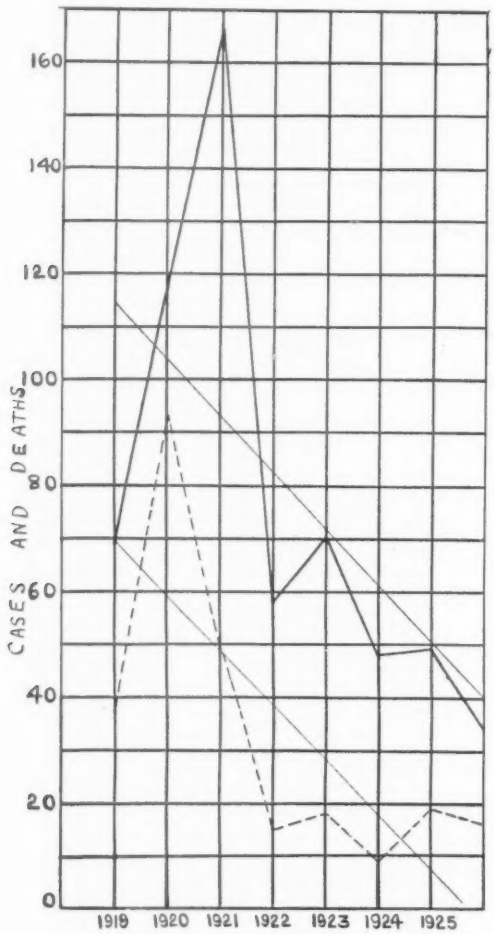

Chart 28. Data collected on encephalitis in Chicago; unbroken line represents number of cases; broken line, number of deaths.

Cook County: Cook County Hospital—1.

City of Chicago: Municipal Tuberculosis Sanitarium, Municipal Contagious Disease Hospital, Isolation Hospital, Iroquois Memorial Hospital, House of Correction—5.

Private Hospitals—98. Total, 107.

Number of Hospitals for Contagious Diseases.—There are three hospitals in Chicago which care for cases of scarlet fever, diphtheria, measles, whooping cough and chickenpox. These are the County Contagious Hospital, the Durand Hospital of the John McCormick Institute for Infectious Diseases and the Municipal Contagious Disease Hospital. The Isolation Hospital cares for smallpox only. The Municipal Tuberculosis Sanitarium, the Chicago Fresh Air Tuberculosis Hospital, the Cook County Tuberculosis Department and the Oak Forest Hospital care for patients with tuberculosis. The Lawndale Hospital is maintained by the Chicago Health Department for the quarantining and treatment of persons with venereal disease. The

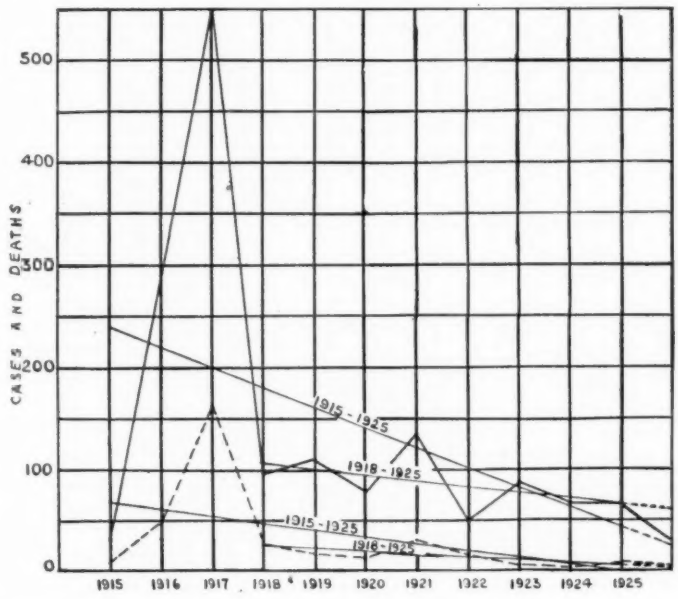

Chart 29.—Data collected on infantile paralysis in Chicago; unbroken line represents number of cases; broken line, number of deaths.

Chicago Health Department also operates two large clinics for the diagnosis and treatment of venereal diseases.

Number of Beds for Contagious Diseases.—In the years 1925-1927, approximately 500 beds were available in Chicago for the care and treatment of patients with contagious diseases other than tuberculosis. It appears that the number of cases of reportable diseases is running approximately 40,000 per annum, declining morbidity balancing increases in the population. On this basis, the number of beds that should be available for patients with contagious diseases should be dictated for the future by present needs and the consideration of whether or not one bed

TABLE 11.—*Hospitals and Hospital Beds in Chicago*

GENERAL HOSPITALS

		Number	Beds, 1925	Beds, 1926
U. S. Public Health	Marine Hospital	1	150	160
State of Illinois	University of Illinois Hospital	2	50	50
	Illinois Eye and Ear Hospital		50	50
Cook County	Cook County Hospital	1	3,000	3,000
City of Chicago	Municipal Tuberculosis Sanitarium	5	900	950
	Municipal Contagious Disease Hospital		270	270
	Isolation Hospital		50	50
	Iroquois Memorial Hospital		25	20
	House of Correction		50	50
Private hospitals		98	5,861	11,526
Total		107	10,406	16,126

HOSPITALS FOR COMMUNICABLE DISEASES

Total Available Beds

	Beds, 1925	Beds, 1926	Diseases
Municipal Tuberculosis Sanitarium	900	960	Tuberculosis
Chicago Fresh Air Tuberculosis Hospital	108	108	Tuberculosis
Cook County Tuberculosis Department	310	310	Tuberculosis
Total	1,318	1,368	
Oak Forest Hospital	594	594	Tuberculosis
County Contagious Hospital	120	120	Scaret fever, diphtheria, measles, whooping cough, chickpox
Durand Hospital	60	60	
Municipal Contagious Disease Hospital	270	270	
Isolation Hospital	50	50	Smallpox only
Total	500	500	

OCCURRENCE OF DISEASES IN VARIOUS HOSPITALS

Hospital Disease	Cases, 1925	Cases, 1926	Deaths, 1925	Deaths, 1926
Cook County				
Scarlet fever	314	147	11	5
Diphtheria	111	181	21	29
Measles	338	175	11	11
Whooping cough	60	41	12	16
Chickenpox	52	85	1	1
German measles	0	16	0	0
Encephalitis	25	2	1	1
Poliomyelitis	28	5	1	0
Epidemic meningitis	5	21	4	10
Erysipelas	234	316	4	30
Typhoid fever	81	71	13	16
Scarlet fever, chickenpox and German measles	0	1	0	0
Durand				
Scarlet fever	621	309	7	5
Diphtheria	261	101	11	8
Scarlet fever and diphtheria	8	2	0	0
Scarlet fever and tuberculous meningitis	1	0	1	0
Scarlet fever and measles	1	2	0	0
Scarlet fever and whooping cough	1	0	0	0
Diphtheria and whooping cough	1	0	0	0
Diphtheria and chickenpox	1	0	0	0
Scarlet fever and German measles	0	6	0	0
Diphtheria, pneumonia and German measles	0	1	0	1
Municipal Contagious Disease				
Scarlet fever	1,549	1,153	23	16
Diphtheria	420	515	51	45
Scarlet fever and diphtheria	142	31	9	5
Measles	1	126	1	3
Whooping cough	6	44	1	5
German measles	26	22	0	0
Chickenpox	8	90	0	0
Encephalitis	1	2	1	1
Poliomyelitis	3	6	0	0
Pneumonia	0	9	0	5
Typhoid fever	0	1	0	1
Diphtheria carrier	0	228	0	1
Mumps	0	12	0	0
Epidemic meningitis	0	3	0	0
Isolation				
Smallpox	89	57	12	2
Chickenpox	5	2	0	0
Oak Forest Tuberculosis Department				
Tuberculosis	...	...	251	236

per eighty cases of communicable disease is sufficient for the normal needs of the community.

Table 11 covers the number of hospitals and hospital beds in Chicago for 1925 and 1926. In addition there is specific information as to the number of beds available for communicable diseases and the hospitals in which such cases are treated in Chicago, with one exception, namely, Oak Forest in Cook County.

Venereal Disease Hospitals and Clinics.—The Chicago Health Department operates two large clinics for the diagnosis and treatment of

TABLE 11.—*Hospitals and Hospital Beds in Chicago—Continued*

TOTAL NUMBER OF COMMUNICABLE DISEASES IN HOSPITALS DURING 1925 AND 1926

Diseases	Cases, 1925	Cases, 1926	Deaths, 1925	Deaths, 1926
Scarlet fever	2,484	2,509	41	33
Diphtheria	792	797	83	101
Scarlet fever and diphtheria	150	33	9	0
Scarlet fever and measles	1	2	0	0
Scarlet fever and whooping cough	1	0	0	0
Scarlet fever and tuberculous meningitis	1	0	1	0
Diphtheria and whooping cough	1	0	0	0
Diphtheria and chickenpox	1	0	0	0
Measles	339	301	12	17
Whooping cough	66	85	13	25
Chickenpox	65	177	1	1
German measles	26	38	0	0
Encephalitis	26	4	2	11
Poliomyelitis	31	11	1	1
Epidemic meningitis	5	24	4	24
Erysipelas	234	316	4	48
Typhoid fever	144	112	29	21
Smallpox	89	57	12	2
Tuberculosis	4,552	3,481	984	1,281
Typhoid Fever				
Hospitals				
Cook County Hospital	81	71	13	16
Private hospitals	63	41	16	5
Patients' homes	92	37	15	3
Total	236	149	44	24
Tuberculosis				
Municipal Tuberculosis Sanitarium	2,779	2,680	236	255
Cook County Hospital (Tuberculosis Department)	1,521	1,582	681	863
Chicago Fresh Air Hospital	252	219	67	57

Evanston		
Evanston General Hospital	250 beds	Medical and surgical
Evanston Contagious Hospital	46 beds	Diphtheria

venereal diseases. In addition, physicians of the health department examine prisoners at the Cook County Jail and the House of Correction. Other agencies operate six large clinics: the Public Health Institute, the Illinois Social Hygiene League, the Central Free Dispensary, the Northwestern Dispensary, the University of Illinois Dispensary and the Cook County Hospital. A number of small clinics are also available. The larger dispensaries and clinics either are free or make a nominal charge. Twenty-six per cent of the cases reported are from agencies under the control of the health department. About the same percentage was reported by one pay clinic. Figures are not available as to the num-

ber of persons applying for examination or treatment at these agencies. On the basis of reported cases, there appears to be a growing tendency for patients to apply to clinics and dispensaries for treatment rather than to private physicians.

The Lawndale Hospital is maintained by the Chicago Health Department for the quarantining and treatment of patients with venereal diseases. Most of the patients are female prostitutes in infectious stages, and they are not discharged until they are no longer capable of spreading the diseases.

While the foregoing agencies are those mainly concerned with this type of communicable disease, there is a growing recognition on the part of large industries and of hotels and restaurants of the importance of employing persons who are free from venereal disease, and there is evidence that prophylaxis is more commonly practiced than was formerly the case.

Expense at Various Hospitals.—The Durand Hospital reports the cost of hospitalization of patients with communicable disease as $3.66 per patient per day in 1925 and as $3.85 in 1926. The Cook County Hospital does not report the cost per day for patients with contagious diseases alone. In 1926 the cost per day for all patients in Cook County Hospital was $3.24. At the Municipal Contagious Disease Hospital in 1925 the cost was $4.06, and in 1926, $4.70.

Cost of Transportation.—It is interesting to record the cost of the ambulance service for patients with communicable diseases in Chicago for the years 1925 and 1926. The cost per patient to be transported in 1925 was $5.60, and in 1926, $9.40.

Results of Hospitalization.—Table 12 shows the total number of patients hospitalized because of communicable diseases, such as diphtheria, scarlet fever, measles and whooping cough, as recorded in the city of Chicago, with the mortality rate in Chicago per hundred thousand population, the fatality rate of cases at the Municipal Contagious Disease Hospital and the fatality rate of all patients who were hospitalized. This special table contains data of extreme importance with reference to the hospitalization of patients in the city of Chicago.

MEASURES BY MUNICIPALITY:

a. PREVENTIVE INOCULATION

Diphtheria.—Since the introduction of vaccination against smallpox, no form of active immunization against a communicable disease in the civil population has received such widespread acceptance by the public and such general endorsement of the medical profession as toxin-antitoxin immunization against diphtheria.

TABLE 12.—*Summary of Hospitalized Cases and Deaths of Certain Contagious Diseases in Chicago from 1918 to 1926*

Year	Disease	Total Cases Reported in Chicago (a)	Total Cases Hospitalized (b)	Total Patients Hospitalized in Municipal Contagious Disease Hospital	Total Deaths Reported in Chicago	Total Deaths of Patients Who Were Hospitalized	Total Deaths of Patients Who Were Hospitalized in Municipal Contagious Disease Hospital	Fatality Rate Total Reported of Chicago	Fatality Rate of Hospitalized Patients	Fatality Rate of Patients in Municipal Contagious Disease Hospital	Mortality Rate of Chicago per 100,000 Population	Patients per Cent Hospitalized at Municipal Contagious Disease Hospital	Deaths per Cent Hospitalized Occurring at Municipal Contagious Disease Hospital
1926	Scarlet fever	4,948	1,372	1,153	79	33	15	1.6	2.9	1.3	2.6	84	45
1925	Scarlet fever	7,668	2,095	1,549	128	58	20	1.7	3.7	1.3	4.3	74	35
1924	Scarlet fever	4,953	1,532	1,022	74	27	6	1.5	1.8	0.6	2.6	67	22
1923	Scarlet fever	3,529	1,079	640	82	31	8	2.4	2.8	1.3	2.9	59	26
1922	Scarlet fever	3,898	1,119	546	110	61	13	2.8	5.5	2.4	3.9	49	21
1921	Scarlet fever	5,459	1,409	623	180	64	9	3.2	4.5	1.4	6.4	44	14
1920	Scarlet fever	8,557	1,331	561	180	43	7	2.1	3.2	1.3	6.6	42	16
1919	Scarlet fever	4,550	.....	356	116	40	12	2.6	...	3.4	4.4	..	30
1918	Scarlet fever	1,809	.....	186	46	17	4	2.6	...	1.9	1.8	..	24
1926	Diphtheria	2,540	1,015	515	224	101	43	8.8	9.9	8.3	7.3	51	43
1925	Diphtheria	2,926	983	535	240	105	64	8.2	10.7	11.9	8.0	54	61
1924	Diphtheria	3,672	1,158	751	218	90	34	5.9	7.7	4.5	7.4	65	38
1923	Diphtheria	5,833	1,949	1,479	362	170	104	6.3	8.7	7.0	12.6	76	61
1922	Diphtheria	7,367	2,378	1,895	558	230	142	7.6	9.7	7.5	19.9	80	62
1921	Diphtheria	9,313	2,621	2,165	683	227	141	7.3	8.7	6.5	24.3	83	62
1920	Diphtheria	7,761	1,974	1,552	626	200	113	8.1	10.1	7.3	23.1	79	57
1919	Diphtheria	6,361	2,375	1,597	592	235	150	9.2	9.9	9.4	22.1	67	64
1918	Diphtheria	5,708	.....	2,090	723	296	242	12.6	...	11.6	27.1	..	82

In a study of more than 100 of the larger American cities, Armstrong and Walker [7] found that nearly all were doing some toxin-antitoxin immunization among school and preschool children. Most of this work, however, has been done among children of school age. Only five cities of those reporting had secured immunization of 25 per cent of the preschool population; 42 per cent of the cities had secured immunization of 25 per cent of the school children. So generally accepted has toxin-antitoxin become that the Appraisal Form of the American Public Health Association gives full credit for control of diphtheria only to those cities which have secured the immunization of at least 25 per cent of their preschool population.

In Chicago, immunization by toxin-antitoxin was begun in 1918. Since 1921, approximately 14,000 children have completed immunization. Practically all of the work has been done in school clinics in the kindergarten and the first grade. Immunization is offered to children of preschool age in the ten infant welfare stations of the Bureau of Child Welfare on Saturday mornings.

TABLE 13.—*Deaths from Diphtheria, and Mortality Rates per Hundred Thousand by Age Groups*

	Deaths			Rates		
Year	Under 5	5–9	10 and Over	Under 5	5–9	10 and Over
1921.....	409	199	68	146	75	31
1922.....	352	162	47	123	60	21
1923.....	212	111	42	73	40	18
1924.....	123	61	32	42	22	14
1925.....	123	88	29	41	31	12
1926.....	117	77	30	39	26	12

One of the striking features in the Chicago mortality statistics of the past few years is the remarkable decline in the death rate from diphtheria. From 24.3 in 1921, the mortality per hundred thousand has fallen to 7.5 in 1926. A similar decline in diphtheria mortality has occurred in a number of large American cities during this period, and in New York the health department has attributed it to the immunization of school children by toxin-antitoxin.

To try to determine whether toxin-antitoxin might be a contributory factor to the decrease in Chicago, the mortality rates from diphtheria have been tabulated by age groups in table 13.

Since practically all immunization done with toxin-antitoxin in Chicago has been in the kindergarten and first grade, it can be considered, for purposes of comparison, that all immunized children are in the 5 to 9 year age group. What effect, if any, has this had on the decline of diphtheria in the various groups?

7. Armstrong and Walker: Immunization Practice in American Cities, Am. J. Pub. Health **16:** 1099 (Nov.) 1926.

In the first place it will be noticed that the decline in mortality has been most rapid in the group under 5 years old, less rapid in the group between 5 and 9 years of age, and least marked in the group over 10 years. The trends in the respective groups are: —23.3, —9.9, and —3.5. There is no evidence that any special factor has influenced the decline in the 5-9 year group.

Since less than 5 per cent of the children in that group have been immunized, however, measurable results could hardly be expected, even if protection in the immunized children is complete. The fact, nevertheless, that as rapid a decline in the mortality of diphtheria in the various age groups occurred in Chicago as in many cities that attributed a similar decline to immunization with toxin-antitoxin should make one critical of unanalyzed figures on the causative relation of that procedure to the falling rate of mortality in diphtheria. No examination of the morbidity of diphtheria in Chicago by age group is possible, because the reports of cases have not been recorded by comparable groups.

Although figures are not available to indicate the value of toxin-antitoxin in Chicago, there is ample evidence of its efficacy when immunization has been extended to a considerable proportion of the population. In Auburn, N. Y., a city of 36,000, where 85 per cent of the school children have been immunized, Sears [8] reported that in three years the number of cases had been reduced from eighty-five to five a year, and deaths from about fifteen to one a year, and that one a questionable case of diphtheria.

An intensive study of some 8,000 children immunized in Providence [9] showed that approximately 90 per cent of the children who received three injections of toxin-antitoxin were subsequently protected against diphtheria. As few of these children were retested, it is probable that such cases as did occur were in children who failed to acquire immunity in one course of injections. Among 15,000 children immunized or naturally negative to the Schick test, the prevalence of diphtheria has been only one-tenth as great as in the same age group of the rest of the population.

If toxin-antitoxin is to play an important part in the control of diphtheria, it is self-evident that a considerable proportion of the children in the more susceptible age groups must be immunized. The ideal procedure would be the immunization of the preschool group, i. e., children aged from 1 to 4 years. Immunization at this time would carry the child over the period of highest fatality and greatest susceptibility to

8. Sears, F. W.: Can Diphtheria be Eliminated? Am. J. Pub. Health. **15:** 98 (Feb.) 1925.

9. Scamman, C. L., and Pope, A. S.: Diphtheria Immunization in Providence, J. A. M. A. **88:**563 (Feb. 19) 1927.

diphtheria. On account of the administrative difficulty in reaching children of this age, it is questionable whether large cities will be able to secure the immunization of a sufficient proportion of this population to control diphtheria.

Immunization of pupils in the kindergarten and first grade leaves unprotected the children at the ages in which the highest fatality per case of diphtheria occurs, but from the administrative standpoint this is probably the most practical scheme.

If any considerable proportion of the school children are immunized, there is also a definite reduction in the exposure in the home of younger children who are themselves less likely to come in contact with the disease. No one has yet shown just what proportion of the population needs to be immunized to prevent any serious spread of a disease of low infectivity, such as diphtheria, but it can be seen offhand how rapidly the increase of immune persons decreases the opportunity for effective contact between susceptible persons.

One of the serious obstacles to securing immunization against diphtheria in Chicago has been the requirement of a preliminary Schick test, although all immunization has been among children under 7 years of age. During 1926, less than two thirds of the children who received the Schick test and the first two treatments came back for the third injection.

Another drawback to the requirement of a prior Schick test is the shortage of personnel to complete the extra work involved. Last year, for this reason, only five sixths of the children for whom consents had been received could be immunized.

On the recommendation of pediatricians and administrators most widely experienced in this field, the Chicago Health Department is arranging to drop the requirement of a preliminary Schick test and to concentrate its efforts on the immunization of more children.

All children immunized are requested to return after not less than six months for a Schick test to determine whether immunity has been established. Exact figures are not obtainable, but a high proportion of retests has been secured.

In Chicago, diphtheria toxin for the Schick test and toxin-antitoxin are supplied without charge by the state, both to the public clinics and to private physicians. The 1/10 minimum lethal dose preparation of toxin-antitoxin is supplied.

Conclusions: 1. Immunization by toxin-antitoxin is of proved value in the control of diphtheria and, as an administrative practice, is well established in most large American cities.

2. The ideal time for immunization against diphtheria is from the first to fifth years of life. On account of the difficulty of reaching the

children in the preschool group, much more has been accomplished in the immunization of children in kindergarten and the first grade school.

3. In comparison with other cities Chicago has been backward in securing protection of its child population against diphtheria. Hardly 5 per cent of the children from 5 to 9 years old have been immunized, and practically none below kindergarten age.

4. Although there is no evidence that toxin-antitoxin has been an important factor in the marked decline of diphtheria in Chicago for the past six years, there is every reason to believe that if immunization is extended to a considerable proportion of the children in the most susceptible age groups, it can be made the deciding factor in the ultimate control of diphtheria.

5. More educational work is needed to convince the public of the value of toxin-antitoxin and to create a demand for general immunization of children at the most susceptible ages.

6. Sufficient funds and personnel should be provided to complete the immunization already requested.

Measles.—Active immunization against measles is still wholly theoretical. Tunnicliff [10] and Ferry and Fisher,[11] following the isolation of what they believed to be the causative organism of measles, prepared a toxin which they reported gave positive skin reactions in persons susceptible to measles and which was neutralized by convalescent measles serum. An attempt to immunize susceptible persons with such a toxin has not been reported.

On account of the universal, high susceptibility to measles of persons who have not had the disease, the persistence of that susceptibility throughout life and the usually benign character of measles after the first three years of life, it seems unlikely that active immunization of a type suitable for general application will be developed in the near future.

The possibility of immunizing susceptible children to measles with the blood serum of patients convalescent from the disease was first demonstrated by Nicolli and Conseil in 1918, and in this country by Richardson and Connor in 1919. Degwitz, and more recently Park and Freeman,[12] have obtained protection in from 85 to 90 per cent of susceptible persons exposed when the serum was given before the fifth day after exposure to measles. In some cases the disease was modified when convalescent serum was given as late as the seventh day.

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10. Tunnicliff, Ruth: Further Studies on the Diplococcus of Measles, J. Infect. Dis. **37**:193 (Sept.) 1925.

11. Ferry, N. S., and Fisher, L. W.: Measles Toxin, J. A. M. A. **86**:932 (March 27) 1926.

12. Park, W. H., and Freeman, R. G., Jr.: Prophylactic Use of Measles Convalescent Serum, J. A. M. A. **87**:556 (Aug. 21) 1926.

The passive immunity conferred by convalescent serum protects against infection for from two to four weeks. In modified measles, a lasting immunity is secured.

Since the discovery of what is believed to be the causative organism of measles, several antiserums have been prepared. Hoyne and Gasul,[13] using an antiserum prepared by Tunnicliff by the injetcion of goats with cultures of a green producing diplococcus isolated from patients with measles, immunized thirty-nine susceptible infants and children at the Cook County Hospital. Of these, thirty-four were protected against measles, and five developed a modified form of the disease. Of the nine not immunized, eight developed measles.

A serum prepared from horses, immunized with the toxins of a streptococcus isolated from the blood serum of patients in the acute stage of measles by Ferry and Fisher, has been supplied to the health department for experimental purposes.

Although the prophylactic value of convalescent serum, given early, is generally recognized, its use has been limited on account of the practical difficulty of providing an adequate supply. Only 12 per cent of the American cities reporting on the matter have taken any steps to supply convalescent measles serum to practicing physicians. In Chicago, convalescent serum is used to a limited extent in the Durand, the Sarah Morris and the Municipal Contagious Disease Hospitals, and is rarely furnished by these institutions to private physicians. In the hospitals its use is limited to infants and to children exposed to measles who are already suffering from some other infection.

As a general practice, it is doubtful whether the immunization of all children exposed to measles is desirable. The passive immunity conferred lasts only from two to four weeks, and the susceptibility to measles is so universal that temporary immunity simply postpones the date of attack. The most successful method of reducing the mortality from measles has been to postpone the age of attack until after the age of 3 years, or at times when children are already weakened from some other infection; and, in accomplishing that end, convalescent serum or a dependable antitoxin is of great value.

Recommendations: In the present stage of development of prophylaxis, it is desirable: (1) that further carefully controlled studies be made, in hospitals and institutions, of the efficacy of the various antiserums for measles now available; (2) that, pending the outcome of such studies, a moderate supply of measles convalescent serum be secured and made available for use by institutions and practicing physicians in the treatment of selected patients who have been exposed to measles.

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13. Hoyne, A. L., and Gasul, B. M.: Measles Prophylaxis, J. A. M. A. **87:** 1185 (Oct. 9) 1926.

Scarlet Fever.—In Chicago, immunization against scarlet fever has been employed only by a few institutions for children and, to a limited extent, by private physicians.

The health department has not attempted any general immunization of children against scarlet fever as against diphtheria. Toxin for the Dick test, or for immunization, is not furnished by the department.

In their survey of immunization practice in American cities, Armstrong and Walker [7] found that 23 per cent of ninety cities which reported recommended active immunization against scarlet fever as a general practice; 28 per cent of these cities furnished toxin for the Dick test and for immunization. None of the cities gave any report on results obtained in the establishment of immunity.

The New York City Health Department, in a personal communication, reported that about 15,000 children were immunized against scarlet fever. The majority of these were in children's institutions. Scarlet fever toxin has not been given out for the immunization of children in the public schools.

Immunity seems to be established quickly and to be effective for a limited time; but, with the dosage given, Doctor Park believes that in children with a strongly positive Dick test it cannot be depended on for more than a year, or a year and a half. At present, four injections, one week apart, of 600, 2,400, 6,000 and 12,000 skin test doses of toxin are used. In some of the Baby Health Stations the scarlet fever toxin and toxin-antitoxin have been given together. Figures are not available on the results of such immunization.

One of the most convincing tests of the efficacy of active immunization against scarlet fever is the experience of the Durand Hospital. From 1913 to 1923, before the introduction of the Dick test and immunization with scarlet fever toxin, of 512 pupil nurses in training forty, or 7.8 per cent, contracted scarlet fever. From Jan. 1, 1924, to Dec. 1, 1927, of 268 pupil nurses in training three, or 1.1 per cent, contracted scarlet fever. Furthermore, none of these three cases developed among nurses whose reaction to the Dick test was originally negative, or those immunized to a negative Dick reaction before entrance on duty.

To show that this reduced incidence of scarlet fever was not due to an atypical form of the disease, without rash, the prevalence of sore throats and tonsilitis among the nurses before and after the introduction of immunization was tabulated. From 1915 to 1923 the incidence of sore throat and tonsilitis was 17 per cent, while for the period from 1924 to 1927 it had fallen to 12.4 per cent.

The procedure of immunization recommended by the Dicks consists of five injections of 500, 2,000, 5,000, 20,000 and 80,000 skin test units of scarlet fever toxin at intervals of seven days. With this dosage,

susceptible persons usually develop a negative skin reaction within from two to three weeks of the last injection.

It is still too early to speak positively of the duration of such conferred immunity. Of the persons immunized at the Durand Hospital many have continued to give negative reactions to the Dick test at the end of the second year. Retests are being made at the end of each year to determine this point more accurately.

At the present stage of development, active immunization against scarlet fever is of distinct value in the following conditions: (1) in institutions for children where complete testing and immunization are possible—here immunization is especially valuable on account of the short time required to develop immunity; (2) among interns, nurses and attendants in hospitals for contagious diseases; (3) in the private practice of pediatricians when full cooperation of the parents can be secured.

Much progress has been made toward securing a practical protection against scarlet fever, but several objections must be overcome before the procedure can be put on the same basis as immunization by toxin-antitoxin against diphtheria. The principal drawbacks to immunization with scarlet fever toxin now are: (1) the short duration of the immunity conferred—one to two years; (2) the necessity of four or five injections to produce immunity. This makes it especially difficult to secure the complete immunization of the children treated and a retest to be sure that they are protected.

Conclusions: Active immunization against scarlet fever cannot yet be recommended as a general public health measure, principally on account of the large number of injections required and the short duration of the immunity conferred.

Active immunization is useful as a control measure in boarding schools or institutions for children in which scarlet fever exists, especially if there is likely to be continued exposure to the disease.

As a prophylactic it is especially valuable for nurses and others exposed to scarlet fever in hospitals for contagious diseases and for the general immunization of children in institutions.

Typhoid Fever.—The efficacy of immunization against typhoid fever with vaccines of killed typhoid bacilli, in populations exposed to infection, is too well established to require discussion here. It seems pertinent, however, to consider the extent of its use in Chicago and its place in a balanced city program for the control of typhoid fever.

The vaccination of persons directly exposed to typhoid fever was begun in Chicago in 1916. Since 1918, all persons who have been in direct contact with patients having typhoid (with the exception of a few women advanced in pregnancy and nursing infants), who have had an exposure less than eighteen days before, have been immunized. For the

past five years, from 1,000 to 1,300 persons annually have been vaccinated against typhoid. Of these, about two thirds were in direct contact with patients in homes; the other third were chiefly nurses and interns in hospitals.

Typhoid immunization of doctors, nurses and attendants brought in contact with patients with typhoid in hospitals is required by the rules of the Illinois Department of Health. In Chicago this rule is enforced in hospitals which treat patients with typhoid. Such immunization could, with advantage, be required of the interns and nurses in all hospitals in the city, and of laboratory workers and students who are likely to be exposed to typhoid fever in their work. The occurrence of three cases of typhoid in laboratory assistants during the past year emphasizes the need of specific protection in workers exposed to that risk.

The Chicago records of 1926 indicate a definite value in the immunization of members of a family who have been in contact with typhoid fever. Of some 500 persons who had been in contact with the disease, fourteen developed typhoid fever. Of these, only four had completed the course of immunizing injections before becoming ill. Since nearly all persons directly exposed who had not already developed typhoid fever were immunized, a control series is not available for comparison. Also, it is known that in part of the series of persons who developed typhoid, immunization was not attempted because the patients already showed symptoms of the disease. Discounting these facts, one must admit that a secondary attack rate of less than 3 per cent indicates more than ordinary success in control of the infection. In his studies of typhoid fever in Washington, Rosenau [14] estimated that in 1908, 15 per cent, and in 1909, 17 per cent of all cases were due to contact infection

In the Chicago data Wright's hypothesis of a negative phase following inoculation is not confirmed. In fact, there is a good deal of internal evidence that the immunity of typhoid is built up quickly enough to be a significant factor in checking the spread of infection.

The efficacy of vaccination against typhoid fever in civil populations has been clearly demonstrated. In a study of certain French towns after the war, Archard [15] found that only 30 per cent of the patients with typhoid fever were males, where before the war approximately two thirds of the patients were males. He also found that such cases as occurred in vaccinated patients were of a milder type than those among the unvaccinated.

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14. Rosenau, M. J.: Preventive Medicine and Hygiene, New York, D. Appleton & Co., p. 116.

15. Archard, C.: Typhoid in Civil Population Since the War, Lancet **1**: 745 (April 15) 1922.

Chauffard and Sargent, in a similar study, found the same change in the sex ratio of typhoid cases after the war, and noted that what typhoid did occur in males was chiefly in those under the age of 20.

The advisability of general immunization in a civil population against typhoid fever depends, obviously, on the prevalence of typhoid in the community in question. In Chicago, with a morbidity of only five per hundred thousand, general immunization of patients of even the more susceptible age group would necessarily pay a small return on the investment.

Since the occurrence of paratyphoid fever in Chicago is about 5 per cent of that of typhoid, all vaccines should contain paratyphoid A and B in addition to *B. typhosus*. War as well as civil experience indicates that a saline suspension, in three doses, is the optimum form of administration.

b. SCHOOL INSPECTION

Medical inspection for communicable diseases in schools is assigned to the school and field health officers of the department of health. Each health officer shall visit the schools in his district daily, beginning at 9 a. m. If there are several small schools in his district, he may, with the approval of the health department, visit such schools regularly on a schedule.

At the opening of school in September each health officer is furnished with a list of all cases of communicable disease in his district, and, on the basis of this notification, he must, on the first day of school, exclude from school all persons and pupils coming from premises in which there is infection unless they have complied with the rules which permit them to reenter school. A rapid inspection of pupils for evidence of communicable disease is required at the reopening of schools and after the Christmas vacation.

Patients in whom a disease is suspected on the preliminary inspection are given a more thorough examination. Cultures are taken from the nose and throat of all children who show sore throats suspected of being infectious, discharging noses or clinical symptoms of diphtheria. Pupils in an infectious condition are promptly excluded from school, and each pupil so excluded is given a notification slip for the parent, stating the cause of exclusion. When patients with reportable communicable diseases are excluded, they are at once reported by telephone to the health department, so that they may be assigned promptly to inspectors for the establishment of quarantine.

As soon as this preliminary examination of the pupils at the schools is completed, the regular morning inspection for communicable diseases is taken up by each health officer, according to his schedule, a copy of which is in each school attended by the health officer. The department and the health officer's superior also each have a copy.

All pupils absent from school one or more days, those entering school for the first time and those received on transfer must be examined by the health officers before they are admitted to school. In pupils with throats in which there is evidence of infection, sore throats, a history of sore throat or nasal discharge, cultures must be made. Principals, teachers, school dentists and nurses are requested to send to the health officer all children suspected of having a communicable disease. At many schools a separate room is provided for the detention of children who are waiting for examination by the health officer. Recently, the department of health recommended to the board of education that children absent from school be requested to return at the hour the health officer is scheduled to make his visit. This should eliminate unnecessary waiting and reduce the danger of spreading infection in the detention room.

In these school inspections the following diseases are looked for: scarlet fever, diphtheria, measles, German measles, mumps, whooping cough, smallpox, chickenpox, tuberculosis, infantile paralysis, epidemic meningitis, influenza, tonsillitis, trachoma, pediculosis, ringworm, impetigo contagiosa, favus and other communicable diseases.

In favus, ringworm, impetigo, scabies and pediculosis, the exclusion is for a period of twenty-four hours only. After being excluded for these diseases, the child on his return to school is examined by the health officer. He is then, if under treatment, readmitted and, if not under treatment, is reported by the health officer to the health department. Such patients are then referred by the health department to the visiting nurse association to see that they receive proper treatment in the home. In cases of pediculosis the children are given, in a sealed envelop with the exclusion slip to the parent, a circular of instructions for treatment of the condition.

When a case of communicable disease is found in a school, the health officer at once notifies the principal in writing of the case. When necessary, the health officer makes further investigation in the homes.

Children recovered from scarlet fever, diphtheria, typhoid fever, smallpox, chickenpox, measles, whooping cough, poliomyelitis or other placardable diseases shall not return to school until the termination notice has been received by the principal from the health department.

Diphtheria.—Whenever a case of diphtheria occurs in a school, the health officer must make a daily inspection of all pupils in the room attended by the patient, for a period of seven days from the last day the patient was in school. In making these inspections the health officer shall take cultures on each inspection from all pupils in whom the appearance of the nose and the throat is suspicious.

Pupils from whom positive nose or throat cultures are obtained must be at once excluded and reported to the health department by telephone,

either as having diphtheria, or as being diphtheria carriers. Pupils who show clinical evidences of diphtheria must be excluded when cultures have been made. All cultures taken shall be obtained from both the nose and the throat.

Immunization against diphtheria with toxin-antitoxin is performed in the schools by the school health officer under an arrangement similar to that for vaccination against smallpox. In the case of diphtheria in a school, immunization is offered to children in the kindergarten and first grade only. Details of the work done and results accomplished are given in the section on active immunization.

Scarlet Fever, etc.—When a case of scarlet fever, measles, chickenpox, whooping cough, German measles or mumps occurs in a school pupil, the health officer must make a daily inspection of all pupils in the schoolroom attended by the patient for the maximum incubation period of the disease from which the patient is suffering, dating from the last day the patient was in school. Any new cases of communicable diseases found by the health officer in making these examinations are promptly excluded and reported to the department of health, as previously described.

Smallpox.—When a case of smallpox occurs in a school, or in the family of a pupil attending school, the principal of the school, on notification from the department of health, excludes from such school for a period of eighteen days from the last probable exposure to such case all pupils and teachers who cannot show definite proof of a successful vaccination within five years or who refuse to be vaccinated. Such exclusion does not apply to pupils or teachers who consent to vaccination as soon as the exposure is known of, or who submit a certificate of vaccination within a reasonable time.

When a case of smallpox occurs in a neighborhood, the board of health may declare an impending epidemic and authorize the exclusion of unvaccinated pupils and teachers according to the provisions outlined previously. This method of procedure has been sustained by the Illinois courts.

School Vaccination.—Annually, the school nurses obtain from each school in their respective districts a list of the names and addresses of all pupils in the kindergarten and first grade of such schools. Inspection for vaccinal status is made by the physician in the kindergarten and the first grade only. Consent cards for the parents' signature are then given to all children who do not show a vaccination scar. Children in the higher grades are advised that if they have not been vaccinated previously or have not been vaccinated within five years, they can get consent cards from the nurse and be vaccinated by the school physician, if the parents' consent is obtained.

All vaccinating in schools is done by the school tuberculosis physicians. These school tuberculosis physicians vaccinate all children for whom consents have been given, including revaccinations in children vaccinated five years or more previously. Reinspection of all vaccinations and revaccinations must be performed within a week. At that time certificates of vaccination are issued to all children who show a typical take. In case of revaccination, certificates are issued after one or two retrials. Not more than two retrials with potent lymph are required if the pupil had a good vaccination scar. The department of health orders revaccination of any and all pupils at any time when epidemic conditions make such action advisable.

The technic used in school vaccination is as follows:

(*a*) The hands of the physician must be sterilized as for other surgical operations.

(*b*) The arm of the patient must be cleansed with green soap and water, followed by alcohol, and dried.

(*c*) The vaccine is deposited at the proper site on the cleansed dry skin and the surface covered by the drop of lymph is scratched with a sterile needle, pains being taken to avoid the drawing of blood. The area scarified shall not be more than one-sixteenth of an inch in diameter. Only one site of innoculation is used.

(*d*) After vaccination, a temporary sterile shield is applied to the arm.

(*e*) The school physician must give explicit instruction to each child to remove this shield not later than the evening of the day on which the vaccination is performed.

(*f*) The school tuberculosis physician must report in writing to the chief of the bureau of child welfare, all cases of infection or very sore arms, following vaccination.

Disinfection of Schoolrooms.—At the request of the department of health, the board of education will disinfect, by washing, sunning and airing, schoolrooms in which cases of communicable disease have recently occurred. In practice, such requests are seldom made. Several days would necessarily elapse between the occurrence of the case and the subsequent disinfection, so that any possible spread of infection from that source would already have taken place. Furthermore, the dissemination of infection from such inanimate objects as dishes and chairs is negligible compared with that from actual cases or carriers of the disease.

Recommendations: 1. The number of school health officers should be sufficiently increased to permit more thorough examination of children in the schools.

2. Special efforts should be made to secure the immunization against diphtheria with toxin-antitoxin and vaccination against smallpox of all children in the kindergarten and first grade in all schools each year.

3. An educational staff should be provided to conduct a campaign to assist in securing the cooperation of physicians and parents for immunization with toxin-antitoxin of children in kindergarten and first grade and at preschool ages.

4. A sufficiently large staff should be provided to perform immunization on all children in these grades for whom consent can be obtained.

5. Practicing physicians should be urged to assume their rightful share of responsibility in the community for the control of communicable diseases, especially in the preschool group of children, and to make special efforts to secure immunization of their patients against smallpox and diphtheria before they reach school age.

c. MILK SUPPLY

The citizens of Chicago consume approximately 1,500,000 quarts of milk per day, giving a per capita consumption of 1 pint to each person per day. The milk to supply this tremendous demand is obtained from 35,000 dairy farms located throughout the states of Illinois, Wisconsin, Indiana, Michigan and Iowa. Approximately 90 per cent of the milk is delivered by the farmers to 150 country receiving stations, where it is chilled to a temperature slightly above freezing. The greater part of the milk is transported into the city from these stations in insulated, glass-lined tanks in refrigerator cars or trucks. The remainder of the milk is shipped in cans directly to the city, by auto truck or train.

All milk, except that which is certified, is pasteurized. Milk for the wholesale trade is pasteurized, to a large extent, in the country plants and shipped to the city in sealed cans. Bottled milk for homes, stores and restaurants is pasteurized almost entirely in the city in 225 pasteurizing plants. Two thirds of 1 per cent of the total milk supply is raw, certified milk produced under the direct supervision of the Chicago Medical Society Milk Commission.

Outstanding recent accomplishments with relation to the milk supply in Chicago are:

1. The adoption and effective enforcement of the requirement that all milk sold in the city must be obtained from healthy cattle, free from disease, as determined by veterinarians testing under state and federal supervision.

2. The elimination of defects in milk plants in the equipment and methods to insure efficient, positive pasteurization with resulting protection against milk-borne infection.

The system of supervision established to safeguard effectively the Chicago milk supply is extremely thorough. It is carried out by a total force of sixty-three civil service employees comprising the Bureau of Dairy Products. Thirty-three country dairy inspectors are constantly engaged in the inspection of the farms, visiting each farm at least once

every six months, for the purpose of insuring that the sanitary conditions under which the milk is produced are satisfactory. Five inspectors of country plants are continuously visiting various receiving and pasteurizing plants, making inspections of equipment and methods and collecting samples for sediment, reductase and bacterial tests, as the milk is delivered from the farms.

The names of farmers and the weight of milk received from each, are entered on lists which are sent in to the department of health. The results of tests are entered on these lists and farmers are advised of unsatisfactory tests, in order to give them an opportunity to improve conditions. On further violation, the farm is shut off until satisfactory assurance is received that the milk will be produced in a clean, wholesome manner.

Each list is checked by trained clerks to determine that a certificate is on file with the department showing that the cattle of each farmer have been tested with tuberculin under state and federal supervision.

The supervision of the milk after it reaches the city is carried out by twenty men. Samples of milk are collected from all dealers for bacteriologic, chemical and sediment examinations. Inspections are made of all pasteurizing plants and milk depots on an average of once each week. With the intensive drive during the year 1926, the percentage of illegal samples of market milk was reduced 35 per cent.

The elimination of defective pasteurizing equipment, started in 1926, has been carried to completion to insure positive pasteurization. A campaign has been started to secure improvement in the location, arrangement and operation in plants not considered entirely satisfactory. This program has already brought about the construction of many new plants and the remodeling of others to give entirely satisfactory conditions for milk plant operations.

Objectives established for the coming year include the practical elimination of illegal samples of market milk; the proper cooling and grading up of the raw milk supply to insure an improved quality of raw milk before pasteurization, and further improvements in the location and arrangement of a number of the city milk plants.

d. WATER SUPPLY

General.—The water supply for the city of Chicago is obtained from six intake cribs in Lake Michigan, located from two to four miles off shore along the Chicago lake front. The water is conducted from these intakes to the eleven municipal pumping stations through approximately sixty-eight miles of underground tunnels. The older tunnels were constructed through clay and lined with brick, the bore ranging from five to ten feet in diameter. The new tunnels are drilled through rock and are lined with concrete, the diameters varying from 10 to 16 feet.

From the pumping stations the water is distributed to the consumers through about 3,400 miles of cast iron pipe varying in size from 4 inches to 48 inches in diameter.

The Chicago water works pumps more water than that of any other municipality in America, and its per capita consumption is considered excessive. During 1925 the total population supplied was 3,241,324, the average daily pumpage during approximately 865,000,000 gallons,[16] equivalent to a per capita consumption of 286 gallons.[17] This is due to the fact that house services are not metered and also that there is underground leakage. A universal metering ordinance was passed in September, 1925. During 1925, approximately 29 per cent of the total consumption was metered, the sale of which water was approximately 58 per cent of the total receipts.

Quality of the Water Supply.—In general, the quality of the untreated public water supply, measured by modern standards, is only fair, depending largely on the intake crib from which the water is obtained. The average bacterial quality of the water from the Wilson Avenue, Carter Harrison and Four Mile cribs is reasonably good. Disinfection, using moderate amounts of chlorine, is practiced. The water from the Dunne and Sixty-eighth Street crib intakes is exposed to a rather heavy degree of pollution during and following periods of prolonged southerly winds. In order to render this water safe, chlorine in amounts varying from 3 to 6 pounds [18] per million gallons is necessary. The average chlorine dosage in pounds per million gallons pumped at all stations during 1924 was 3.3; 1925, 3.61, and 1926, 3.57.

The chlorine is applied at the stations in the suction well as near to the supply tunnel as is practical. As Chicago does not have any reservoir system, the period of contact between the chlorine and the water is relatively short, with the result that it is necessary to carry a higher chlorine dosage than would otherwise be required. This period of contact varies from approximately twenty seconds to two minutes, depending on the station and the rate of pumpage.

At each of the pumping stations the chlorinating equipment is in duplicate to provide against emergencies. A chlorine attendant is on duty day and night at all stations and gives his full time to operating the chlorine equipment and making hourly tests for residual chlorine to permit a rigid control. A residual chlorine content of at least 1 pound per million gallons is required in the water as delivered to the distri-

16. The daily consumption for 1927 was approximately 900,000,000 gallons.

17. For 1927 the per capita consumption averaged about 300 gallons per day.

18. On Dec. 28, 1927, during a period of extreme polution by sewage and industrial wastes, it was necessary to use 13.2 pounds of chlorine per million gallons to safeguard the public health.

bution system, and the operators at the stations are permitted to increase, but not to decrease, the quantity of chlorine to maintain this minimum residual.

The responsibility for prescribing the quantity of chlorine used for disinfection rests with the Division of Water Safety Control, while the actual application of this chemical comes within the jurisdiction of the Division of Pumping Stations Operating, both divisions being in the Bureau of Engineering, Department of Public Works. The Division of Water Safety Control inspects the operation of the chlorinators at the pumping stations and reviews the records of the previous day. Besides control of chlorination this division is also charged with the responsibility for all matters relating to the safety of the public water supply. These include investigations of sources of pollution of the lake waters near the intakes; investigations of leakage into tunnels; cross-connections in the distribution system, and numerous other minor matters.

Daily samples of the unchlorinated and chlorinated water are collected at representative sampling points in the tunnel system and at the pumping stations and are delivered to the laboratory for the analysis of water in the Department of Health. Approximately 15,000 bacterial analyses of the public water supply are made annually for routine control purposes. In addition, daily analyses of the water for turbidity and content of plankton are made in these laboratories, along with monthly sanitary chemical analyses. Records of cases of typhoid fever are kept by the various water districts and any unusual developments are promptly investigated. It will be seen, therefore, that the activities for the control of water safety are carried out on a cooperative plan between the departments of health and public works.

Sources of Pollution of Water Supply.—There are five outstanding sources of pollution of the public water supply:

1. The most important source of pollution is the sewage and industrial wastes discharged from the Indiana Harbor Ship Canal, which is approximately 8.5 miles south of the Sixty-Eighth Street and Dunne cribs. Following periods of prolonged southerly winds pollution from this source arrives at the cribs and jeopardizes the quality of the public water supply.[38] The industrial wastes, which frequently contain compounds of phenol combine with the chlorine used for disinfection and impart an objectionable "medicinal" or "carbolic" taste to the water on the south side of the city.

2. The polluted waters of the Calumet River near its mouth discharge into the lake approximately 40 per cent of the time. The Calumet light opposite the mouth of the river is 3¾ miles due south of the previously mentioned cribs.

3. The Illinois Steel Company's Eightieth Street sewer, with a 6-foot outlet, is 2½ miles south to southwest of these cribs and the wastes which are discharged are obtained from the plant's grossly polluted North Slip, being first used for hydraulic and condensing purposes.

4. Sewage and wastes are deposited by freight and passenger vessels arriving at and departing from this port.

5 The lake waters are polluted by the dumping of dredged and excavated material into the lake.

Exclusive of these sources of pollution, which are due to human factors, the water supply of Chicago would still be more or less unsatisfactory as a first-class product because of the natural tubidity in the lake water following violent storms and also because of tastes and odors caused by the plankton growths normal to Lake Michigan. The periods of highest turbidity are generally in the winter and spring months. The highest turbidity of recent years occurred in December, 1926, being 200 parts per million for two days. The characteristic grassy to fishy tastes and odors due to plankton begin in July and persist with more or less intensity through September.

Filtration for Improvement of Quality.—The solution of the problem as to the quality of Chicago's water lies in filtration. At present, a plant for the experimental filtration of water is being designed and will be constructed at the Sixty-Eighth Street pumping station before July 1, 1927.[19] This plant will cost approximately $150,000 and will permit extensive studies in water filtration. On the basis of the information obtained, plans will be adopted for the design of large filter plants for the entire city. It is probable that the first municipal filtration plant will be built to serve the south side of Chicago.

The filtration problem is closely allied to the water wastage situation and until such a time as the excessive wastage can be overcome the indications are that it will be uneconomical and impractical to build a filter plant. It is hoped, however, that the meter installation program will be developed at the time the filtration plant designs are completed.

Sanitary Control of Swimming Pools and Bathing Beaches.—In Chicago there are approximately sixty indoor public swimming pools, natatoria and plunges; thirty-three outdoor bathing and wading pools in public parks; three municipal bathing beaches where public bath houses are operated, and a large number of street and public beaches at which millions of people bathe each year. In general, with the exception of the first-class clubs and private institutions, supervision over the quality of bathing water is inadequate. Equipment for proper recirculation and disinfection of the water is woefully lacking in many institutions, and control is unsatisfactory in others which have this equipment. In general, the need for improvement of sanitary conditions in the public swimming pools of Chicago is acute. With proper recirculating systems and equipment for disinfecting the water, a great improvement in conditions could be effected.

19. This experimental plant was placed in operation in March, 1928.

The outdoor bathing pools in the public parks are popular and their bathing load during the summer months is usually heavy. In general, recirculation and disinfection are not practiced, dilution being depended on for maintaining a satisfactory bacterial quality of the water. There is opportunity for much improvement in the operation of these pools.

The bathing beaches in Chicago are popular, and in general the quality of the water is reasonably satisfactory. A rather intensive survey conducted during 1924 indicated that dilution and wave action in the lake resulted in maintaining a reasonably good quality of water at these beaches. It is true that where the bathing load is exceptionally heavy, especially in shallow waters, the contamination is usually heavy. This is difficult to control without restricting the number of bathers.

Some of the bathing beaches on the extreme south side are exposed to pollution from the Illinois Steel Company's sewer and the Calumet River. Bathing in the Chicago and Calumet rivers is prohibited, and is not generally practiced, although minor violations occur.

EDUCATION OF MEDICAL STUDENTS

The medical student generally makes his first academic acquaintance with communicable diseases in a course in pathogenic bacteriology which he is required to take, either in his first or second year. A small, but increasing, proportion of medical students have had some bacteriology as part of their college curriculum, and some have had general college courses dealing with problems in public health, but relatively few enter the medical school with the required amount of bacteriology. The course in bacteriology in the medical school is almost exclusively a pre-clinical course. While the student becomes familiar with the characteristics of the pathogenic micro-organisms and with the laboratory methods of clinical bacteriology, he rarely is given the opportunity of seeing cases of communicable disease. In the clinical years of his course in the medical school, he receives further instruction in contagious diseases through didactic lectures and instruction in the class room. By arrangement with the Chicago Department of Health, each medical student in the four large medical colleges in Chicago receives one week's instruction at the Municipal Contagious Disease Hospital. At this time he sees such cases as are available and also obtains some laboratory experience in diagnosis. Three hospitals offer opportunity for intern service: the Durand Hospital, the Municipal Contagious Disease Hospital and the Cook County Hospital. In the latter, work in the wards for contagious diseases is part of the rotation service. Aside from the opportunities here described, the medical student sees only casual cases of contagious diseases.

EDUCATION OF NURSES

In order to determine what measures were being taken to give nurses training in the care of communicable diseases, letters were sent to the superintendent of the training school of Cook County, Durand, Mercy, Michael Reese, Presbyterian and St. Luke's Hospitals. The following replies were received:

Cook County Hospital.—Laura R. Logan, R. N., dean of the Illinois Training School for Nurses: "Our students receive their practice in the care of contagious diseases in the Cook County Contagious Hospital on the grounds of Cook County Hospital. We have supervisors who are especially prepared to do the bedside teaching and we use the technic used at Durand Hospital for Contagious Diseases. Miss Charlotte Johnson, who is in charge of Durand Hospital, assists in the teaching of our student nurses. Members of the medical staff of Cook County Hospital assigned to Contagious Hospital assist in the teaching, Dr. Archibald Hoyne carrying most of the work. We also extend study and practice to a number of affiliated schools in Illinois and other midwestern states.

"Student nurses receive immunization before going to Contagious Hospital through the Doctors Dick or their assistant, Dr. Rhodes, who is our school physician.

"I believe very thoroughly that every student in every school of nursing should have the opportunity to become thoroughly prepared in the nursing care of contagious diseases. I should be glad to cooperate with the Illinois League of Nursing Education or the Chicago Institute of Medicine in any movement to promote proper standards of nursing education and service in our City Contagious Hospital in order to extend as widely as possible the benefits of the experience which could be made available there. There are many schools of nursing in Chicago which offer no training whatever in contagious disease nursing. This is a deplorable situation."

Mercy Hospital—Sister Mary Lidwina, R. N.: directress of nurses: "I regret to inform you that our students do not get any practice in this work.

"Since you requested any suggestion on the matter, frankly speaking I think that nurses wishing to specialize in this field should take a special course in contagious nursing after graduation."

Michael Reese Hospital—Dora C. Saunby, R. N., principal School of Nursing: "Our students are offered three months of communicable disease nursing at Durand Hospital as an elective course in their senior year. This provides experience for only two students each three months. The training which they receive is of the highest character both from the standpoint of technic and their development preparatory to assuming the responsibilites of a graduate nurse. Durand Hospital is very small and does not offer an adequate field for a city the size of Chicago. In spite of the fact that we need a larger field, this work must be developed under exceptionally good conditions or our students could not take advantage of it."

Presbyterian Hospital.—M. Helena McMillan, R. N., director, School of Nursing: "Student nurses of this school have the privilege of three months' work in contagious diseases at the Durand Contagious Hospital under the supervision of Miss Charlotte Johnson, superintendent. We are entirely satisfied with the experience and teaching they receive there. Miss Johnson is thoroughly qualified both by experience and personality to be in charge of a group of student nurses."

Durand Hospital—Charlotte Johnson, R. N., superintendent: "There are one hundred forty-five accredited schools for nurses in the state of Illinois. Five thousand six hundred twenty-three students were enrolled in these schools during 1926. All these institutions give a course of theoretical instruction in communicable diseases. Student nurses are occasionally assigned to the care of patients suffering from acute infections in the general hospital wards where a more or less careful technic is carried out.

"Two general hospitals have departments for communicable diseases and give practical training only to their own student nurses. Three hospitals caring for acute infectious diseases offer courses in practical and theoretical instruction to students from outside accredited schools.

"As nearly as can be determined less than 200 or about 3.5 per cent of the student nurses enrolled in schools for nurses of Illinois last year obtained from one to three months' experience in the actual bedside care of patients suffering from acute communicable diseases. From these revealing statistics we must admit that this is a seriously neglected field of nursing. There is need for an awakening to a deeper sense of community responsibility on the part of members of hospital boards, superintendents of schools for nurses, staff physicians and people generally who are in any way interested in the preparation of the nurse for usefulness to society.

"From the standpoint of the welfare of the patient as well as the safety of the community the highest type of skill and most intelligent service are required in the care and management of acute infectious diseases. The nurse who has not had adequate preparation in medical aseptic nursing is no better prepared for her task if called upon to establish efficient isolation and to give intelligent nursing care than the student on her first day in the operating room is prepared to direct the exacting technic required there. 'Practice makes perfect' is an old trite saying which today is as true as ever. No amount of theory can take the place of practice in the acquirement of any art or skill whether it be music or golf or nursing. It is unfair to the young graduate, to her patient and to the public to expect her to give satisfactory service in this exacting line of duty without adequate practice under careful supervision.

"Many registered nurses refuse to care for patients suffering from acute transmissible diseases for two reasons: first, because they have not had sufficient experience in medical asepsis and realize the danger of faulty technic both to themselves and to others and are unwilling to assume the serious responsibility of caring for acutely ill patients on account of their lack of familiarity with symptoms, conditions and technical care required; second, because they feel the hardship of the rulings of the department of health at the present time which shut the nurse up with the patient, giving her no opportunity for the freedom and exercise necessary to her safety and physical well being.

"When schools for nurses strengthen this weak link in the chain of their requirements in the standard curriculum, when health officers are convinced that nurses not only clearly understand the principles of aseptic nursing but can actually apply them practically and efficiently, simpler and less burdensome quarantine regulations will be in force. So long as the health officer doubts the ability of the nurse to carry out strict medical asepsis, he feels he must protect the community from the danger of the weaker members of the nursing group.

"It is unfortunately true that medical asepsis has lagged far behind surgical asepsis. What surgeon would undertake an operation without first removing his coat, rolling up his sleeves, putting on a clean gown and scrubbing his hands? How many medical men are as consistently careful when coming and going from

patients suffering from communicable diseases? Medical asepsis is simple, so simple in fact that essential details are easily overlooked. It might be defined as precise personal cleanliness. Early childhood is the best time to plant this ideal. Certain important personal habits should be taught the young child old enough to understand that he should keep his fingers and other things which may be contaminated, out of his mouth, that he should wash his hands before eating, and after soiling his fingers with mucus or other things. School children, especially eighth grade and high school girls should be taught simple isolation measures in preparation for the safety of themselves and others during epidemics.

"Young mothers should be instructed as to the importance of keeping the sick child with his own equipment in his own room away from other members of the family. She should understand the danger of contamination by fresh moist discharges. She should know the danger of becoming a 'carrier' herself and the necessity of avoiding kissing and close personal contacts either with her sick or well child. She should be carefully taught how to remove infectious material from her hands. She should be instructed to put on a gown or suitable garment *always* before caring for her sick child and should be warned *never* to leave the sick patient's room without first removing this gown and cleansing her hands thoroughly. Each member of her family should have at all times, separate towels, toilet articles, etc. Any intelligent mother with a clear understanding of the dangers to be avoided can, within a short time under careful guidance, be taught how to care for her convalescent child safely when he is no longer acutely ill and has little or no discharge from his mucous membranes, and at the same time prevent infectious materials from being spread to other members of her family.

"Careful medical asepsis should be taught to every medical student and student nurse during the first days of their contacts with patients. Adequate facilities for cleansing the hands should be available within easy reach of every bedside. The hands should *always* be washed after coming in contact with *any patient* before touching another. This may seem like a 'counsel of perfection' but even so the old surgeon of fifty years ago would class our surgical technic of the present day. If preventive medicine is the goal ahead, all available forces should be marshalled in the conquest of disease."

RECOMMENDATIONS

1. The committee wishes to call attention to the specific recommendations presented in the special contributions of Alton S. Pope, H. O. Jones, G. W. Putnam, A. E. Gorman and Charlotte Johnson on the subjects of preventive inoculations, school hygiene, milk supply, water supply and the nursing of patients with communicable diseases, respectively.

2. The committee does not feel justified in making specific recommendations at this time on increased hospitalization of patients with communicable diseases.

The splendid results obtained at the Municipal Contagious Disease Hospital show what may be accomplished by the efficient administration of an official agency.

The committee feels strongly that all hospitals should provide facilities for the care of patients with communicable disease. All hospitals

should be expected to provide facilities for the care of patients who contract or develop a communicable disease while under observation or treatment for other ailments. Hospitals must recognize that they have moral as well as financial obligations in these matters, however much they may object to retaining patients with communicable diseases because of the expense of the special nursing care required by existing regulations of quarantine.

3. The problem associated with regulations of quarantine and their modification is of such importance and magnitude that the committee recommends the appointment of a special committee to study and report on this problem.

QUARANTINE

The following is an abstract of the revised rules and regulations of the Illinois Department of Public Health for the control of communicable diseases effective July 1, 1927:

Disease	Quarantine of Case and Contacts	Exclusion from School
Chickenpox	Case. Seven days or until all scabs have disappeared from skin.	Case. Until quarantine is terminated, or until all scabs have disappeared from the skin.
	Contacts. No restrictions.	Contacts. Not excluded from school.
Diphtheria	Case. Minimum period 16 days, or until two successive negative cultures are obtained from nose and throat. Terminal cultures may be taken 24 hours after the membrane has disappeared from the nose and throat and the temperature is normal.	Case. Until termination of quarantine and two successive negatives have been obtained from the nose and throat.
	Contacts. At home. Kept under quarantine with case. Discharged on one negative culture when case is terminated. If removed to other premises, kept under observation until two successive negative cultures are obtained from the nose and throat.	Contacts. Until one negative culture is obtained from the nose and throat of each contact.
	Case. From 4 days before and until 5 days after the appearance of the rash and thereafter until all catarrhal symptoms have ceased.	Case. Until recovery and until the cough and other catarrhal symptoms and abnormal mucous membrane secretions have ceased.
	Contacts. Susceptible contacts for 14 days from date of last exposure.	Susceptible Contacts. Fourteen days from last exposure.
Mumps	Case. For seven days from the onset of the disease and until all glandular swelling has disappeared.	Case. For seven days from the appearance of the disease and until all glandular swelling has disappeared.

Disease	Quarantine of Cases and Contacts	Exclusion from School
Scarlet Fever	Contacts. No restrictions. Case. Twenty-eight days and until throat is normal and all discharges from the ears and suppurating glands have ceased. Contacts quarantined with case are kept under quarantine until termination of the case. Susceptible contacts away from home—quarantined for 7 days.	Contacts. Not excluded. Case. For 35 days or until the throat is normal and all discharges from the ears and suppurating glands have ceased. Contacts who reside on the infected premises excluded until one week after termination of quarantine. Non-immune children who have been exposed and do not live on premises must be excluded for 1 week from last exposure.
Smallpox	Case. Minimum period of three weeks and until the skin is entirely smooth and the "seeds" have disappeared from the palms of the hands and soles of the feet. Contacts. Kept under quarantine for 16 days unless immune by virtue of an attack of smallpox or by reason of a successful external vaccination within five years.	Case. For a mininum period of three weeks from onset of eruption and until the skin is smooth and the "seeds" have disappeared from the palms and soles of the feet. Non-Immunes. For 16 days from last exposure.
Whooping Cough	Case. Until three weeks after the beginning of the whoop and until paroxysmal cough has disappeared. Susceptible Contacts. Ten days from last exposure and extend if cough develops. Immunes. Not quarantined.	Case. Until after expiration of quarantine period. Contacts. Susceptibles must be excluded for 10 days from date of last exposure. Immunes. Not excluded.
Epidemic Meningitis	Case. Quarantined for two weeks after onset of the disease. Contacts quarantined with case must remain until termination of quarantine. Contacts away from quarantined premises quarantined for 10 days.	Case. For one week after termination of quarantine. Contacts. Excluded for 1 week after termination of quarantine. Contacts. Living elsewhere excluded for 10 days.
Polio (Infantile Paralysis)	Case. Three weeks from date of onset until all abnormal discharges from nose have ceased. Contacts quarantine with case until termination of quarantine. Contacts (under 16) living elsewhere are quarantined for 14 days from last exposure.	Case. Until one week after termination of quarantine. Contacts. Non-immunes excluded for 2 weeks and immunes for 1 week from date of last exposure.
Typhoid Fever	Case. Until 2 successive negative specimens of feces and urine are obtained. Specimens to be collected not less than 24 hours apart, the first specimen to be taken 10 days after normal temperature.	Case. Excluded from school until released from quarantine. Contacts. None required, provided there is no contact with the patient.

Disease	Quarantine of Cases and Contacts	Exclusion from School
	Contacts may go about their usual business, unless they are engaged in the handling of food, foodstuffs or milk. All contacts should be immunized with typhoid vaccine.	
German Measles	Case. Eight days from appearance of rash. Contacts. No restrictions.	Case. Eight days from the appearance of the rash. Contacts. Not excluded.

Pediatric Biographies

LEONELLUS FAVENTINUS VICTORIUS
? — 1520

GEORGE KHUFNER, Jr.
? — ?

JOHN RUHRÄH
BALTIMORE

Victorius, also known as Vettari or Vettorio, was born in Faenza in the Romagna. He studied medicine in Bologna, and after qualifying for practice, settled down to a physician's life in the same city. Not much is known about the details of his life, but in 1473 he became a professor in the university, where he taught logic, philosophy and medicine. On his retirement from his professional duties, he settled in Feltre in the Province of Belluno, where he died in 1520. His practice and teaching were much influenced by the writing of the Arabian physicians. He wrote two books, one about children and the other about the practice of medicine. The latter was published at Ingolstadt in 1545, and bears the title, "Practica medicinalis. Liber de medendis morbis membrorum omnium totius corporis humani, nunquam antea in lucem editus. Hoc opus novum et antehac nullibi excusum Joannes Kufnerus Trochoreus brevibus scholiis illustravit (Ingolstadtii, 1545)."

The original edition of the little book on the diseases of children was printed in Ingolstadt in 1544, and was reprinted at Lyons in 1546, 1554 and 1574; the Venice edition, listed by Meissner, is dated 1557. The first edition is extremely rare, and Victorius is best known through the later reprints, which were the work of George Khufner. The second edition is entitled, "De aegritudinibus infantiu tractatus. Appendix a eundem, per Georgium Khufnerum juniorem. Quibus accesit oratiuncula, per eundem Georgium Khufnerum publice habita."

It is not clear whether Khufner thought the work of Victorius especially worthy of preservation for succeeding generations or whether he thought to introduce his own book more quickly and successfully by tacking it on to the work of an older writer. In any event, few pediatricians would possess a copy of Victorius had it not been for Khufner.

Victorius' book consists of thirty-three short chapters, each on a common disorder of childhood. He first names the disease, and after a

LEONELLI FAVENTINI DE VICTORIIS,

De ægritudinibus infantiū Tractatus.

Appendix ad eundem, per Georgium Khufnerum iuniorem.

Quibus acceſsit Oratiuncula, per eundem Georgium Khufnerum publicè habita.

LVGDVNI,
Apud Antonium Vincentium.
1546

few lines of comment, gives the "cure." It is more a compendium of infantile therapeutics than a textbook of the diseases. He was evidently a great believer in external remedies; perhaps he found them safer in infants and children. He uses fomentations, inunctions, plasters, baths, powders, oils, unguents, liniments, embrocations and sternutatories. If he did not help the child much, neither did he hinder the course of nature a great deal, for according to John Locke's dictum, "It is better to do nothing than to do amiss." He was a wise physician, at least, as far as therapeutics goes. The following excerpts on pain in the stomach is an example of his work:

ON PAIN IN THE STOMACH

Pain in the stomach occurs in infants sometimes from flatulence existing in the intestines, sometimes from the coolness of the atmosphere or some other cause chilling the stomach; sometimes also it results from worms biting the intestines.

CURE

This ailment therefore is cured: First, by rubbing the child's stomach with hot water and fomenting with a sponge. For it opens the pores of the stomach and breaks up the flatulence.

It is effective for the same purpose to take common oil, to which should be added a modicum of crocus, and in this oil immerse rags and apply them to the stomach.

FOMENTATION FOR THE SAME

℞ meliloti, chamomillae ana M ss.
anisi, foeniculi, carui ana M ss.
Let all boil in half a measure of water until half has boiled away, then let the place, *i. e.*, the stomach, be fomented with a sponge three or four times. Afterwards let the place be anointed with the ointment described below.

OINTMENT

℞ olei chamaemelini, liliacei, anethini ana 3 ss.
Mix, let an anointing be made, as above.

CURE

The cure of the pain arising from worms is to be had in the cure of worms.

Khufner added sixty-one chapters of his own, which are not unlike those of Victorius. He covers a wide range, from the diseases of the hair, teeth, skin and anus to diseases of the heart, epilepsy, hernia and numerous other disorders.

In the following extract on diabetes, it may be noted that when he did not know the cause he gave one anyway, a practice dear to the medical writer and as much in use in the present scientific age as when Khufner wrote.

ON EXCESS AND LACK OF URINATION, AND FIRST ON DIABETES

The diabetic disease is an excessive and involuntary flow of water. Its cause is excessive heat of the kidneys, gently absorbing the wateriness of the blood.

The symptoms are: Immoderate and unnatural flow of urine; indeed whatever is drunk, quickly passes out in the raw state, and frequently the urine is white and clear because of its rawness and indigestion.

Prognosis: This disease brings on extreme emaciation, and unless it is attended to in time, dropsy and death are to be feared.

Cure: Milk prepared from filberts recently broken, in a confection of water of urinative herb, is wonderfully beneficial to all diabetics. Galen recommends barley water, given to drink, and *rob fructum.*

Among the simple remedies (are) ends of vine, quinceapples, pomegranates, grass of willows, purslane, shepherd's staff and evergreen, taken separately or mixed, after the manner of an abscess as well as of fomentation or bath.

Plaster for the same

℞ farinae hordeaceae ʒ iij

Let it be infused in oil of rose q. ss. and a modicum of vinegar. There may also be added purslain seeds, a shaving of cucumber, and solatrum, with the addition of cooling seeds, small as well as large, cleansed of their bark; and let a plaster be made according to the art.

News and Comment

MEETING OF AMERICAN ASSOCIATION FOR THE STUDY OF GOITER

The American Association for the study of Goiter will meet in Denver, Colo., June 18 to 20.

SOUTHERN PEDIATRIC SEMINAR

The Southern Pediatric Seminar meets at Saluda, N. C., July 23 to August 4 under the deanship of Dr. W. A. Mulherin, with Dr. Frank Howard Richardson as vice dean and D. L. Smith as registrar. This seminar is carried on by the best pediatricians of the South and is purely an act of altruism, since not even traveling expenses are paid to any one of them.

DEATH OF DR. ROSS SNYDER

Dr. Ross Snyder of Birmingham, Ala., professor of pediatrics of the Post-Graduate Department of the University of Alabama and pediatrician to several Birmingham hospitals, a well known pediatrician of the South, died recently of pneumonia.

APPOINTMENTS AT YALE

Yale University announces the following appointments to the Medical Faculty on May 6: Dr. Joseph I. Linde and Dr. Donald W. Porter, as professors of pediatrics; Dr. John W. Hammond and Dr. Daniel C. Darrow, as assistant professors of pediatrics.

THE NEW CHEVALIER JACKSON BRONCHOSCOPIC CLINIC OF THE NEW GRADUATE HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA

Announcement is made by Dr. George H. Meeker, dean of the Graduate School of Medicine of the University of Pennsylvania of the opening on March 31, 1928, of the newly and completely equipped Chevalier Jackson Bronchoscopic Clinic of the new Graduate Hospital of the University of Pennsylvania at 19th and Lombard Streets, Philadelphia.

The new bronchoscopic clinic will be in personal charge of Dr. Chevalier Jackson, professor of bronchoscopy and esophagoscopy in the Graduate School of Medicine of the University of Pennsylvania.

Abstracts from Current Literature

Anatomy and Physiology

RESEARCHES ON THE OCCLUSION OF THE FONTANEL IN NORMAL AND DISEASED STATES. MOURIQUAND, BERNHEIM and LACAUX, Arch. de méd. d. enf. **30**:27, 1928.

The relation existing between ossification of the anterior fontanel and the general nutrition of the infant has been the subject of numerous observations, but the data thus obtained have not always been satisfactorily presented. With the object of rendering the knowledge of this condition more precise, the authors have undertaken an intensive study, continued over a considerable period. Their first study was the consideration of the fontanel in healthy infants: (*a*) its dimensions at birth and throughout the intervening months between this period and complete ossification; (*b*) the normal time for closure. Basing certain conclusions on these studies, they proceeded to the investigation of abnormal infants, eliminating such disorders as cerebral conditions, encephalopathy, hydrocephalus and mongolian imbecility. In other cases, notably of myxedema, in which the hypothyroidism was acute and the fontanel was tardy in closing, they argue that the latter phenomenon may properly be added to the tests for endocrine insufficiency. Special inquiry was made in those cases presenting manifestations of rickets, however slight. Here the results conflicted with the opinion usually held among pediatricians that frank instances of this disease commonly show considerable delay in the ossification of the fontanel. Of 105 cases, 70 showed normal occlusion, in spite of the marked rachitic lesions. Among these, 56 infants had been breast fed and 14 artificially nourished. The monograph brings out the value of mother's milk in encouraging normal development of the bones.

The relation between delay in bridging the cranial sutures and delay in eruption of the teeth was also carefully studied, and parallelism was not found to exist.

AMESSE, Denver.

STRIDOR THYMICUS. I. ROTH, Monatschr. f. Kinderh. **38**:223 (Jan.-Feb.) 1928.

Roth reports a case of dyspnea in which a roentgenogram showed an enlarged thymus. Unfortunately, there is no report of the result of roentgen-ray therapy.

ORTHOSTATIC ACIDURIA. E. FAERBER and T. DEMETRIADES, Monatschr. f. Kinderh. **38**:243 (Jan.-Feb.) 1928.

This is the second of the studies made by these authors. They show that the kidney secretion is definitely influenced by the position of the body. In the normal child, the reaction of the urine depends just as much on position of the body as it does on diet. The upright position causes a diminished flow of blood through the kidney, a diminished flow of urine with lowering of the nitrogen and the sodium chloride output, and aciduria. The writers emphasize that these changes take place just as much in normal children as in those of the orthostatic type.

GERSTLEY, Chicago.

ESTIMATION OF PHYSICAL DEVELOPMENT OF CHILD GROUPS. A. D. OSTROVSKY, J. Pediat. (Russian) **2**:225, 1927.

The author discusses the methods of determining the degree of physical development of child groups. He distinguishes two cases:

1. The group in question forms a part of a large geographic group. This group may then be compared with other groups of the same geographic subdivision. The

author advises in such cases not merely the determination of the average value of each characteristic but its evalution in terms of σ, the standard deviation, by the use of the formula of Bunak $\left(\frac{W-M}{\sigma}\right)$, in which W is individual value, M, arithmetical mean, and σ the standard deviation of the varying group).

2. The group in question is not a part of any homogeneous whole. In this case various indexes must be used. The author discusses those proposed for the weight-stature relation $\left(\frac{P}{L} \cdot 100\right)$, $\left(\frac{P}{L^3}\right)$, etc., and shows their inadequacy, especially in giving a systematic increase in index with increase of stature. The author proposes the index $\frac{P}{L^{2,66}}$, the exponent having been determined empirically, and in such a way as to eliminate the "modifying influence of stature."

SEVERAC, Philadelphia.

Pathology, Bacteriology and Biochemistry

THE EFFECT OF GENTIAN VIOLET ON THE ORGANISM OF BLASTOMYCOTIC INFECTION. EVERETT S. SANDERSON and DUDLEY C. SMITH, Arch. Dermat. & Syph. **16:** 153 (Aug.) 1927.

Blastomyces was markedly inhibited in cultural growth by dilutions of gentian violet up to 1:1,000,000. It is suggested that the dye would be found useful in treating blastomycotic infections.

JEANS, Iowa City.

THE ACID BASE EQUILIBRIUM IN THE BLOOD OF CHILDREN. 1. TITRABLE BLOOD ALKALI. K. HILLE, Arch. f. Kinderh. **82:**94 (Sept. 30) 1927.

The first of these studies deals with the effect of bathing on the blood alkali. Rohonyi's method was used, and the bath given was of temperatures varying between 25 and 40 C. It was found that there was a lowering of the blood alkali by hot water, and a slight increase with cold water and indifferent bathing. There was also a lowering in fever and by hyperventilation. It must be remembered that there is considerable individual variation to these results, but the variations of alkaline change in the titrable blood alkali must be of some significance.

CALCIUM AND PHOSPHORUS CONTENT OF THE BLOOD SERUM IN INFANTS. O. ULMER, E. HILLENBERG and P. SCHIMMELPFENG, Arch. f. Kinderh. **82:**179 (Sept. 30) 1927.

The blood serum phosphorus held to a fairly definite curve, averaging about 3.5 mg. when the child had not taken food, and running to 5.8 mg. four hours after food was taken. After six hours it had dropped to 4.5 mg. The calcium in the blood serum averaged about 11.2 mg., and rose to 12.0 mg. after two hours. It had returned to normal after six hours.

THE CATALYTIC ACTION OF THE BLOOD WITH ESPECIAL REFERENCE TO CHILDREN. H. BISCHOFF, Arch. f. Kinderh. **82:**189 (Sept. 30) 1927.

Four hundred and fifty-two cases were studied, the micromethod of Bach and Zubkowa being used. The average normal found in these cases for the catalytic action of the blood was 16.1 for adults, 16.8 for children and 14.1 for infants. This varies as to the capillary position from which the blood was drawn; the ear has a higher value than the heel or finger. In children, there was apparently little change in the catalytic action from heliotherapy or from the usual contagious diseases. There was a slight lowering after food was taken.

SANFORD, Chicago.

PROTEIN THERAPY IN THE LIGHT OF EXPERIMENTAL TESTS. A. WOLFF-EISNER, München. med. Wchnschr. **73**:2114 (Dec. 10) 1926.

Wolff-Eisner calls attention to the fact that protein therapy may simply serve to mobilize antibodies in the organism and therefore is really not an aspecific but a specific therapy.

AMBERG, Rochester, Minn.

TETANY. II: THE EFFECT OF EPINEPHRINE AND ERGOTAMINE IN EXPERIMENTAL TETANY. M. B. BRAHDY and T. BREHME, Ztschr. f. exper. Path. u. Therap. **59**:232 (Jan.) 1928.

The nature of the muscle spasm in tetany was investigated by noting its effect on the lactic acid and dextrose content of the blood before and after the administration of epinephrine and ergotamine. The tetany was artificially produced by hyperventilation. There was an increase in the lactic acid associated with the tetanic spasms. However, even if the spasms were greatly diminished or entirely prevented by the injection of ergotamine, the rise in lactic acid was the same. Alkalosis from oral administration of alkalis resulted in a rise in the lactic acid of the blood. The increase noted during the tetanic spasms was assumed to be due to the alkalosis which occurs during hyperventilation. The muscle spasm in tetany is apparently different from the ordinary muscular contraction, since the latter itself results in a rise in the lactic acid of the blood. The possibility that it may be a special form of muscle tonus is discussed.

AUTHORS' SUMMARY.

SUBCUTANEOUS SALT FEVER. HILDEGARD FRANK and HERBERT POGORSCHELSKY, Ztschr. f. Kinderh. **43**:549 (May) 1927.

In his latest publication, Rietschel states that in the subcutaneous demonstration of saline solution so many factors come into consideration that it is idle to discuss which factor is the cause of subcutaneous salt fever. This led the authors to attempt an investigation of what rôle bacterial contamination may play. It is their conclusion that saline solutions injected in amounts up to 100 cc. do not cause any mechanical injury to the tissue which could produce a fever; that all fever reactions which occur after this or smaller quantities have been injected are to be regarded as a water deficiency fever. One can judge of the significance of salt fever only when hypertonic solutions in fresh, carefully redistilled water cause fever.

FURTHER RESEARCHES ON INTESTINAL BACTERIA AND A CONTRIBUTION TO THE BIOLOGY OF THE BACILLUS BIFIDUS. E. STRANSKY and H. MASLOWSKI, Ztschr. f. Kinderh. **43**:717 (July) 1927.

Studies in the culture of *Bacillus bifidus* show that it grows easily in mediums that are poor in protein but rich in sugar under amebic conditions. In breast-fed infants, the same factors may condition the growth of this organism.

EMERSON, Boston.

Nutrition

DYSTROPHY IN ARTIFICIALLY FED INFANTS. HARRY M. GREENWALD, M. Clin. N. Amer. **11**:1055 (Jan.) 1928.

Dystrophy may be divided into two large groups: "dystrophia simplex," which may or may not be accompanied by diarrhea and in which loss of weight is gradual, and "dystrophia hydrolabilis," characterized by rapid loss of weight and usually diarrhea.

Simple dystrophy may result from any of the following causes: insufficient supply of fluid, relative excess of milk (milk injury), relative excess of carbohydrate (starch injury), lack of accessory foods, excessively dilute

formulas, diets containing insufficient calories and vomiting (pylorospasm). It may also follow parenteral infections, pyelitis, chronic nasopharyngitis, etc., and accompany constitutional disturbances such as cerebral anomalies and congenital heart disease.

The diarrhea occurring in dystrophic infants is of three main types. Fermentative diarrhea may result from the feeding of proper mixtures in amounts exceeding the infant's tolerance. Stasis occurs with upward migration of intestinal organisms, resulting in abnormal carbohydrate fermentation, and the formation of excessive amounts of fatty acids, which irritate the intestinal wall and increase peristalsis. Diets too rich in carbohydrates may produce the same results.

Inanition diarrhea is most frequently found in the hydrolabile new-born as a result of starvation. If food deficiency is qualitative, it is usually the carbohydrate that is lacking.

Irritative diarrhea may be due to infection of the intestinal mucosa with typhoid, paratyphoid or dysentery bacilli, or to toxins from parenteral infections.

The author describes in detail the treatment of the various forms of dystrophy, the correction of qualitative and quantitative deficiencies in the diet, and the adaptation of various foods to the tolerance and caloric requirements of the infant.

Two conditions may arise in dystrophic infants that often are difficult to overcome, namely, vomiting and anorexia. The infants who vomit should be treated by judicious starvation — the subcutaneous use of 10 per cent dextrose and stomach lavage. In mild anorexia, waiting may be advisable. If the condition is severe, the following procedures may be useful: changes in the technic of feeding; addition of from 8 to 10 drops of dilute hydrochloric acid to the formula; lavage; change in type of feeding and, finally, tube feeding.

SMITH, Ogden, Utah.

CITRIC ACID MILK AS A DIET FOR DYSENTERIC CHILDREN. S. MEYER, Arch. f. Kinderh. **82**:241 (Nov. 11) 1927.

Citric acid milk is prepared by boiling whole milk for five or ten seconds. It is then cooled quickly by cold running water. Five grams of powdered citric acid is then added to each liter of milk, the required amount of sugar also being added at the same time. This mixture is constantly stirred, so as to make a fine curd. The caloric value of this milk is between 850 and 900 calories per liter. The acid content does not vary much and is between p_H 4.4 and 4.5.

This milk is a satisfactory food for infants suffering from dysentery. The best results were obtained by giving it with rice gruel.

SANFORD, Chicago.

THE SPECIFIC DYNAMIC ACTION IN VITAMIN DEFICIENCY. A. VON ARVAY, Biochem. Ztschr. **192**:369 (Jan. 26) 1928.

Deficiency in vitamin B results in a diminution of the basal metabolism and almost complete disappearance of the specific dynamic action. Deficiency in vitamin A does not have an effect on either the basal metabolism or on the specific dynamic action. The author thinks the effect of deficiency in vitamin B is due to a diminished activity of the thyroid and hypophysial glands.

BRAHDY, New York.

THE PATHOGENESIS OF DISTURBANCES OF NUTRITION AND OF DIGESTION IN EARLY CHILDHOOD. M. MASSLOW, Ztschr. f. Kinderh. **43**:604 (July) 1927.

Within the limits of the same anomaly of constitution, whether it is asthenic, lymphatic or arthritic, individual variations in the amount of gastric juice are important, so that the figures quoted in this article can be looked on only as characteristic of the general tendency of the organisms.

1. In asthenic children, the gastric juice is relatively rich in pepsin and rennet, though there is considerable fluctuation, great diminution during digestion and incomplete restitution in the amount of gastric juice, characteristics which justify one in speaking of the labile type of stomach. The duodenal juice is poor in amylase and trypsin.

2. In lymphatic children, the gastric juice is rich in ferments, is strongly acid during digestion and the amount and strength are constant. This is the nonlabile type of stomach.

3. Arthritic children show a poverty in gastric ferments and in acids and with good strong trypsin, feeble amylase. These results reported by Professor Masslow of the Military Medical Camp of Leningrad are clarifying in respect to clinical symptoms which are peculiar to one or the other type of constitutional anomaly.

EXPERIENCE WITH IRRADIATED FOOD AFTER EXTRACTION WITH ETHER. EVELINE AYRER, Ztschr. f. Kinderh. **43**:668 (July) 1927.

The diet of McCollum, 3143, subjected to repeated ether extraction, is extremely poor, at best, in stearin. It becomes brown after irradiation for one-half hour with ultraviolet light and is activated and protects young rats against rickets, while animals from the same litter fed with the same but nonirradiated food become severely rachitic. If the protection against rickets is conditioned by remnants in the food of stearin which escaped extraction by ether, it must be assumed that the part of stearin capable of activation contained in wheat and corn is much higher than that in the commercial preparations of cholesterol.

IS THE ANTIRACHITIC ACTIVATION OF MILK CLOSELY RELATED TO ITS CONTENT OF STEARIN? E. EDELSTEIN, Ztschr. f. Kinderh. **43**:681 (July) 1927.

Edelstein thought it would be of interest to establish whether it would be possible to activate milk after cholesterin was separated from it. Two infants with florid rickets were fed for four weeks with half strength milk which was made from skimmed milk powder, irradiated and then subjected to extraction as much as possible with ether. On this food, animals with rickets remained uninfluenced clinically, roentgenologically and by chemical tests of the blood. The addition of 3 Gm. of irradiated cholesterin to the food, otherwise the same, over a period of two and three weeks, caused a definite improvement in the rickets in three or four weeks. The antirachitic activation of milk seems to be bound up with its ether soluble component, and by exclusion it may be assumed that the vehicles of this activation are the stearins. The activatible component of milk stearin is relatively higher than in commercial cholesterin.

EMERSON, Boston.

Prenatal Conditions

TRANSMISSION OF PROTEIN HYPERSENSITIVENESS FROM MOTHER TO OFFSPRING. BRET RATNER, HOLMES C. JACKSON and HELEN LEE GRUEHL, J. Immunol. **14**:249, 267, 275, 291 and 303, 1927.

Critic of Placental Permeability.—The passage of heterologous substances through the placenta is a physiologic, and not a pathologic function. The placenta is permeable to antitoxins, precipitins, bacteriolysins, heterologous proteins, protein sensitizing antibodies, etc., in man, guinea-pigs and rabbits, but not in cows, goats and sheep. Explanation of this difference of permeability is based on histologic differences of placentas in various species: in man and rodentia a single cell membrane separates the maternal and fetal circulations, while in ruminants, three cell layers separate the two circulations.

The Rôle of Colostrum.—Work on the transmission of protein hypersensitivity by the colostrum was not found in the literature. Colostrum is an essential medium for the transmission of immunity from a mother to the newborn in ruminants (goats and cattle), but in rodentia (guinea-pigs and rabbits) and in the human species, the colostrum plays a negligible rôle in this transmission.

The Rôle of Milk.—Evidence was not found of a transmission of sensitizing proteins from the milk of nursing guinea-pigs to the suckling offspring. The literature also discloses a negligible rôle for the milk in the transfer of antibodies to the suckling, except in mice and ruminants.

Passive Sensitization in Utero.—Passive hypersensitiveness to horse serum, acquired in utero by the offspring of sensitized guinea-pigs, occurred with a high degree of regularity. This passive state is due to the transference of hypersensitiveness from the mother through the placenta, and is not hereditary. This hypersensitivity is transmitted to successive litters presumably throughout the period of fecundity of the guinea-pig, and persists for about two and a half months definitely, and occasionally for four months to a moderate degree.

Active Sensitization in Utero.—Definite active sensitization of new-born guinea-pigs can result from the passage of antigen from the mother to the fetus in utero. The close correspondence of the mother-fetus relationship in guinea-pigs with that in the human being therefore allows the hypothesis that infants manifestly allergic to the first contact with a foreign protein have been sensitized in the uterus by the placental passage of antigen from the mother's circulation.

OBSERVATIONS AND RESEARCH ON THE HEMAGGLUTININS OF MOTHER AND FETUS. M. PROSPERO and G. MORRA, Arch. per Sc. méd. **49**:714, 1927.

The blood group of the child is completely fixed and manifest at birth. Fetal iso-agglutinin is not derived from the mother, but develops in the fetus during intra-uterine life and is generally of low concentration at birth. Isolysin is weak and its demonstration difficult. The permeability of the placenta does not affect the blood grouping. No difference of weight was found for children born of mothers belonging to a different group from that of the child.

AUTHORS' SUMMARY.

CONGENITAL ABSENCE OF BOTH INFRARENAL GLANDS? W. VOIGHT, Centralbl. f. allg. Pathol. u. path. Anat. **40**:387, 1927.

A woman, aged 43, whose first child died of convulsions at the age of 10 weeks, whose second pregnancy resulted in a stillbirth and whose third pregnancy resulted in a miscarriage, entered the hospital with the clinical signs of Addison's disease, a strongly positive Wassermann reaction and two indolent ulcers of the left knee. Evidences of status lymphaticus were found at autopsy, although the thymus was atrophic. Both suprarenal glands were missing, as were also their arteries from the usual sources. Small nodules of suprarenal cortical tissue were found adhering to the upper poles of the kidneys, and in some of these were masses of central fibrous connective tissue heavily infiltrated with round cells. The ulcers of the knee were thought to be syphilitic, but there were no other evidences of syphilis in the body. The possible explanations for the observations are: first, that some disease, possibly syphilis, caused atrophy of the suprarenal glands, although in such an event the process must have been marked to destroy completely all vessels; and second, that other chromaffin-possessing cells such as those of the sympathetic nervous system, carotid body, etc., took on the function of the suprarenal medulla but finally failed to supply the insufficiency because of a failure to hypertrophy.

GEORGE RUKSTINAT.

Diseases of the New-Born

ICTERUS NEONATORUM GRAVIS: REPORT OF A CASE WHICH RECOVERED WITH SEQUELAE POINTING TO INVOLVEMENT OF THE CENTRAL NERVOUS SYSTEM. H. M. GREENWALD, Am. J. M. Sc. **174**:793 (Oct.) 1927.

Icterus neonatorum gravis is almost uniformly fatal. The author found in the literature only eleven cases in which recovery and subsequent involvement of the central nervous system were reported. The case reported was the mother's third pregnancy, the first two infants having died of this disease at 5 and 11 days, respectively. In this instance, the jaundice appeared the day after birth and persisted for five weeks. The child maintained constantly a normal weight curve; bleeding and coagulation time were normal. At 6 months, the child was unable to support the head; at 1 year, it could stand when held under the arms, but only on the toes and with legs crossed; at 1½ years, speech was definitely retarded, the appearance was idiotic, the child stood on the toes, gait was spastic and unsteady, the upper extremities were incoordinate and a bilateral Babinski sign was present. The various theories of this disease are discussed, and it was thought that sepsis, maternal toxemia and syphilis could be ruled out as etiologic factors in this case. The nature and extent of the neurologic involvement are reviewed and the prevalence of intellectual deficiency stressed; the author would add this disease as a possible cause of true congenital cerebral diplegia.

KUBITSCHEK, Philadelphia.

PNEUMOTHORAX IN A NEW-BORN INFANT: CASE REPORT. M. J. FLIPSE, J. Florida M. A. **14**:403 (Feb.) 1928.

A well developed girl, weighing 8 pounds (3.6 Kg.), was delivered with low forceps.

Vigorous efforts to breathe were made immediately after delivery, but little if any air entered the lungs. The usual methods failing to induce respiration, a tracheal catheter was introduced and active artificial respiration continued. Partial relief soon followed, and the catheter was removed. Expansion of the chest was unequal, apparently being limited to the left side. Cyanosis occurred on crying. Inhalations of oxygen gave temporary relief. The tenative diagnosis was pulmonary atelectasis. Roentgenograms showed the left lung to be 50 per cent collapsed, with a well defined pneumothorax completely surrounding it. Additional roentgenograms showed a progressive collapse of the lung and increasing pneumothorax. Two days after birth, the left lung was completely collapsed. Thirty cubic centimeters of air was aspirated from the pleural cavity; this gave some relief. The following day, aspiration was again resorted to.

Following this some stridor occurred which soon disappeared, and on the following day the pneumothorax had disappeared.

The etiology of the case was obscure but was probably due to a collapsed epiglottis, and inspiratory efforts at birth were sufficient to rupture an alveolus.

LOVE, Jacksonville.

CONGENITAL MALARIA. LJACHOWETZKY, Centralbl. f. Bakteriol. **103**:380, 1927.

The author analyzes his material obtained from thick blood smears of mothers and their new-born children, and he concludes that a direct transfer of malaria from the mother to the child occurs through the placenta. He points out that in view of this fact, it is necessary in the campaign against malaria to treat pregnant malarial carriers in order to prevent the birth of children who may constitute a new series of malarial carriers.

PAUL R. CANNON.

Acute Infections

TULAREMIA WITH REFERENCE TO ITS CUTANEOUS MANIFESTATIONS. E. W. NETHERTON, Arch. Dermat. & Syph. **16**:170 (Aug.) 1927.

A case of tularemia with secondary skin lesions is reported and the literature reviewed. When cutaneous lesions are present, they are mainly of two types: (1) subcutaneous nodules which simulate those seen in sporotrichosis and along the lymphatics which drain the primary lesion; (2) erythematous papules and plaques which follow exacerbation or relapse of systemic symptoms. These may simulate erythema multiforme or an exanthem.

JEANS, Iowa City.

SPECIFIC PREVENTIVE MEASURES IN DIPHTHERIA, SCARLET FEVER AND MEASLES. JAMES H. TOWNSEND, Boston M. & S. J. **196**:611 (April 14) 1927.

The following conclusions are the result of the author's experience as health director at St. Paul's School.

1. Immunization of all infants to diphtheria with toxin-antitoxin during the second six months of life is strongly recommended.

2. Passive immunization with antitoxin of scarlet fever contacts is not at present to be recommended because of the relatively severe serum reactions which sometimes occur.

Active immunization to scarlet fever with either pure toxin or ricinoleated antigen is recommended to be used in families and in institutions in which the disease has appeared. As a routine procedure it also has a place in institutions, but it is not yet ready for general use until there is more knowledge of the duration of the immunity.

3. Passive immunization to measles with convalescent human serum, or immune goat serum, is a valuable and practical procedure in families and institutions. Active immunization to measles has yet to be developed.

GENGENBACH, Denver.

I. A METHOD FOR ESTIMATING THE POTENCY OF SMALLPOX VACCINE. II. THE IMMUNOLOGICAL RELATIONSHIP OF ALASTRIM AND MILD SMALLPOX. JAMES P. LEAKE and JOHN N. FORCE, Bull. 149, Hyg. Lab., U. S. P. H. S.

The first article contains a review of the literature on potency tests for smallpox vaccine, and an outline of a method for making potency tests. A vaccine to satisfy the criterion of potency outlined should produce in all previously unvaccinated subjects a circular vesicle measuring at least 7 mm. in diameter on the seventh day after application, undiluted to a circle of the exposed derma measuring 2 mm. in diameter.

The second article contains a review of the literature indicating the identity of the variola-like disease with a low mortality, which has prevailed in the West Indies, Brazil, Africa and Australia under various names, with the mild smallpox of the United States and Canada. In the experiments undertaken, the fact that definite cross-immunity exists between alastrim and mild smallpox and between alastrim and vaccine virus is additional evidence of the identity of alastrim and mild smallpox.

PREVALENCE OF DIPHTHERIA IN THE UNITED STATES. Pub. Health Rep. **43**:313 (Feb. 10) 1928.

The preliminary report from thirty-seven states for 1927 shows a reaction to the extremely low mortality from diphtheria in 1926. There is an increase of 16.7 per cent over 1926, and 15 per cent over 1925. The greatest prevalence of diphtheria occurred in October, November and December.

AN INQUIRY INTO THE RELATIONSHIP BETWEEN HOUSING CONDITIONS AND THE INCIDENCE AND FATALITY OF MEASLES. J. L. HALLIDAY, Medical Research Council, London, 1928.

The mechanism is shown by which certain types of tenement buildings may contribute to a high death rate from measles by exposing to the infection a larger number of children at the earlier years than is the case in an area in which the cottage type of house predominates. It is evident that, although measles in Great Britain is essentially a disease of childhood, the period of a child's life in which an attack takes place differs considerably according to the way in which circumstances vary. Among crowded tenement quarters housing members of the working class, such as are to be found in Glasgow, the disease is essentially one of children under school age. In the working class and middle class families of Birmingham, who live in houses of the single-entry type, the disease is predominantly one of scholars of from 5 to 10. In country districts also, the disease tends to be one of school children of from 5 to 10. Among the public school class, the age of attack seems to be postponed to a still later period of school life. It would appear that, taking the case mortality for children from 5 to 10 as equal to unity, the case mortality from measles is:

For ages 0 to 2, from 10 to 20 times greater.
For ages 2 to 3, from 8 to 11 times greater.
For ages 3 to 4, from 3½ to 4½ times greater.
For ages 4 to 5, from 1½ to 2 times greater.

SANFORD, Chicago.

GLYCEMIA IN DIPHTHERIA. LERREBOULLET and PIERROT, Arch. de méd. d. enf. **31**:148, 1928.

In the course of their clinical studies with diphtheria at the Hospital for Sick Children, Paris, the authors have had occasion to investigate the various theories advanced in recent years to account for the alarming chain of symptoms so frequently seen in untreated patients coming late to the hospital and designated by Marfan as presenting the secondary syndrome of malignant diphtheria. Among these symptoms may be mentioned cardiac inhibition through irritation of the pneumogastric, bulbar inflammation, functional disturbance of the suprarenals and renal insufficiency with retention of nitrogen. Numerous writers have stressed the possibility of functional or organic disease of the suprarenals as a factor in the promotion of grave accidents in the course of diphtheria. Their contention is supported by the fact that autopsy frequently reveals extensive hemorrhage in the capsule, and by the additional observation that suprarenal extract has been shown to have had remarkable therapeutic effect in many severe forms of the disease. In collaboration with Marie and Leprat, a report was made (1922) on the variation in blood sugar found in ordinary and in malignant cases of diphtheria. This investigation was prompted by two well established phenomena:

1. The marked hypoglycemia which constantly attends suprarenal insufficiency. This is particularly noted in Addison's disease.

2. Diphtheria, in its gravest forms, is at times characterized by clinical manifestations strongly resembling those found in complete abrogation of the capsulary function.

In addition, animal experimentation with diphtheria toxin demonstrates clearly the frequency of grave lesions in these organs.

Of six patients with malignant forms of the disease in the first series, the authors found five with decided hypoglycemia (from 0.59 to 0.77, the standard being placed at 0.90). They interpreted these symptoms as indicating that a lowered blood sugar in the course of diphtheria represented the harmful effects of the toxin on the suprarenal bodies. In 1926, this study was resumed

with forty children suffering from malignant angina, and the results carefully checked with similar investigations on patients with eruptive fevers. In this work the standards set by the American biochemist, Folin, were rigidly observed. In ordinary cases of diphtheria, the sugar content of the blood was practically normal; in malignant cases it varied from 0.45 to 0.85 Gm.; in two patients with extensive diphtheritic paralysis, the figures were slightly below normal, but in a third patient, treated vigorously by antitoxin and epinephrine, the blood sugar was elevated to 1.35 Gm. In the eruptive fevers (measles and scarlet fever), the glycemia was either normal or elevated. It is argued that the extent of the hypoglycemia indicates the relative damage to the suprarenals and thus constitutes an important element in prognosis. The use of epinephrine in conjunction with serum therapy is strongly advised.

AMESSE, Denver.

BACTERIOLOGIC STUDY OF DYSENTERY IN PORTO RICO. O. C. MANDRY, Porto Rico Rev. Pub. Health and Trop. Med. **3**:259 (Jan.) 1928.

Examination of the stools of seventy-seven patients with acute diarrhea revealed the Flexner type of dysentery bacillus in only six cases. Various other organisms of possible significance in intestinal disorders, including *B. paratyphosus* B, *B. enteriditis,* and *B. suipestifer,* were encountered in both diarrheal and nondiarrheal cases. It is pointed out that in bacillary dysentery the onset is acute with fever and toxemia, pain and tenderness. The stool is liquid and has shreds of mucus and blood, with an abundance of cellular exudate. In amebic dysentery the onset is insidious, without fever. If pain and tenderness are present, they are more localized. The stool tends to retain its fecal character, and has small splashes of blood, with a scantiness of cellular exudate.

SANFORD, Chicago.

CLINICAL NOTES ON RABIES IN CHILDREN. BRUNO TRAMBUSTI, Riv. di clin. pediat. **25**:739, 1927.

A boy, aged 6 years, had been bitten by a dog, receiving a slight scratch on the cheek forty days before the onset of symptoms of rabies. He had received antirabic treatment. He died four days later. An autopsy was performed and the diagnosis established. The pathologic methods of value in the study of the disease are outlined and discussed.

HIGGINS, Boston.

PARATYPHOID IN INFANTS. O. ULMER, Arch. f. Kinderh. **82**:1 (Aug. 5) 1927.

Seven cases of paratyphoid in infants were reported by Gobel in *Arch. f. Kinderh.* (**80**:181, 1927). Ulmer reported two further cases that were typical of this condition.

COMBINED ACTIVE AND PASSIVE IMMUNIZATION AGAINST DIPHTHERIA. H. OPITZ and G. MEYER, Arch. f. Kinderh. **82**:11 (Aug. 5) 1927.

After a thorough study of forty cases in which the treatment was by active and passive immunization, with suitable controls, the authors decided that this method is satisfactory. Passive immunization will often stop the active formation of a protective material, but as the further increase of an already existing antibody is slight, the combination of active and passive immunization against diphtheria may be procured.

PREVENTION AND TREATMENT OF WHOOPING COUGH BY VACCINES AND ETHER. F. VON BERNUTH and P. HANNEMANN, Arch. f. Kinderh. **82**:33 (Aug. 5) 1928.

The authors warn against deep muscular injections in the treatment of whooping cough. In four intergluteal injections of ether, results were not

obtained, and paralysis of the tibial and peroneal nerve zones apparently resulted from such injections.

SANFORD, Chicago.

THE MICROSCOPIC CHANGES IN THE LARYNX IN WHOOPING COUGH. FRANZ HEINRICHSBAUER, Jahrb. f. Kinderh. **118**:104, 1927.

Thirteen larynges from patients with a clinical diagnosis of whooping cough were available for microscopic study. One group of sections were cut from the lateral wall to include the vocal cords and another from the interarytenoid at the posterior commissure, taking in part of the pharynx. Gram stains were made for bacteria.

In four cases, typical ulcerations were found on the vocal cords; in three there was a perichondritis in the arytenoid region, in all cases marked capillary dilatation, and round cell infiltrations about the crypts of Morgagni. In many cases agglomerations of lymphocytes were found, often about efferent lymphatics. In four cases, the mucous membrane about the vocal cords was piled up. In two cases a portion of the vocal cord was superficially necrotic (tuberculosis in one?).

For the ulceration on the cords, the author assumed a primary inflammation from a flapping together during a paroxysm followed by ulceration, a vicious cycle being thereby established and a probable site for instigation of pneumonia. The lesions did not have a specific character.

The other changes were inflammatory and could not definitely be called specific for whooping cough.

TRAUTMAN, Milwaukee.

ETIOLOGY, PROPHYLAXIS AND SERUM THERAPY OF INFECTIOUS DISEASES. KRAUS, Wien. klin. Wchnschr. **40**:149 (Feb. 2) 1928; concluded **40**:200 (Feb. 9) 1928.

The author, who is director of the Vienna state serum institute, reviews his own and other recent investigations in infectious diseases. He believes that the occurrence of postvaccinal myelitis is due to a tendency on the part of modern human beings to neurotropic poisoning. When vaccinations against rabies and smallpox are performed, such accidents can be prevented if a vaccine is employed, the virus of which has been killed by phenol or ether (Fermi's method).

In diphtheria toxin-antitoxin mixtures, according to Kraus, there is always the danger of dissociation of the toxin with subsequent harm to the patient. Ramon's anatoxin (toxin rendered nonpoisonous by the addition of solution of formaldehyde) is a safe and equally efficient substitute. Toxoids have also been produced from tetanus toxin and from the vibrio of cholera.

The author believes that Calmette has adequately demonstrated the efficiency of B.C.G. in the vaccination of infants against tuberculosis. Kraus and others, however, have shown that this attenuated tubercle bacillus is sufficiently virulent to produce transient tuberculous lesions in experimental animals. For this reason, he cautions against the general employment of this method until its safety has been conclusively established by further authoritative clinical and pathologic studies.

He maintains that the scarlatinal streptococcic serums produced by the Dicks and Dochez are fundamentally similar to that which Moser used twenty-five years ago. Recent experiments with the original type of Moser serum have established its specificity. The most important contribution of the Dicks is the skin test devised by them. This furnishes a means of detecting susceptibility to scarlet fever and, in addition, provides the only known method of estimating the potency of the toxin and antitoxin. Kraus suggests that subsequent investigation may prove that scarlatinal streptococcic antitoxin is "paraspecific," i. e., capable of neutralizing more than one strain of streptococci.

With regard to the Degkwitz method of preventing measles by injection of convalescent measles serum, the author holds that central agencies must take charge of securing such serum, if an adequate supply is to be obtained.

In clinical investigations of Besredka's antivirus, he has found it to be analgesic and curative in several infections (phlegmon, angina, purulent peritonitis, tuberculous laryngitis and lupus). The theoretic explanation of its action, which as yet is undetermined, may place it in the group of non-specific therapeutic agents, which includes colon vaccine, foreign proteins and malaria in treatment of syphilis.

Several other advances in the realm of serum therapy are described.

OLMSTED, Seattle.

Chronic Infections

SHOULD PERSONS WITH TUBERCULOUS PERITONITIS BE TREATED MEDICALLY OR SURGICALLY? BRODY, Arch. de méd. d. enf. **31**:5, 1928.

Ever since 1862, when Spencer Wells operated on a patient for ovarian cyst and found tuberculous peritonitis instead, which fortunately was cured, the latter disease has been definitely classified as surgical. The result of operative procedures has been so uniformly good that few could object to them. Unfortunately, grave accidents and complications have accompanied some of these operations, among which may be noted fecal fistulas of rebellious character. Following the observations of such distinguished surgeons as Alglave, Berard, Hartmann, Tussau and Le Fort, there is now a decided reaction from the former attitude and clinicians generally are inclined to differentiate certain types of tuberculous peritonitis from the category of surgical diseases. For example, operative intervention is contraindicated in the acute forms and in the ulcerocaseous varieties with adhesions. Instead of indiscriminate laparotomy, surgeons are now recommending a preliminary trial of the ultraviolet ray or of heliotherapy. If operation is considered imperative, Temoin suggested that it be done in the sunlight. In addition to combined phototherapy and surgery, vaccines and serums have been employed in the treatment of this condition. Autohemotherapy, peritoneal paracentesis, pneumoperitoneum, diathermy, radium therapy, continuous drainage and the roentgen ray have all been used with more or less satisfaction. The author believes that every case has individual peculiarities; that these should be studied carefully before any line of treatment is decided on, and that one should bear in mind the remark of Peter that "the belly is still waiting for its Laennec."

AMESSE, Denver.

THE PROPHYLAXIS OF HEREDITARY SYPHILIS. G. DUJOL and C. LAURENT, Presse méd. **35**:1553 (Dec. 2) 1927.

Two hundred and thirteen women were treated during pregnancy with intravenous arsenicals. In 161 pregnancies, 92 per cent of the children were alive at three months. There were 0.4 per cent of macerated fetuses, and 2.5 per cent of miscarriages. The still-births amounted to 5 per cent.

This was a vast improvement over the results of former pregnancies of the same women. In the former cases 26 per cent of the children were living at three months. Twenty-five per cent were born dead or died before three months, and there was 34 per cent of miscarriages, with 15 per cent macerated fetuses.

Subcutaneous injections of pentavalent arsenic was used in treating infants. Twenty of the twenty-two treated developed normally.

SANFORD, Chicago.

THE ETIOLOGY OF SECONDARY TUBERCULOUS MENINGITIS. ROBERT DEBRE and ROBERT SENOZE, Rev. franç. de pédiat. **3**:830, 1927.

In this article Debre, who has already contributed much to the understanding of tuberculosis in childhood, and his co-worker M. Senoze, have paid particular attention to the frequency with which tuberculous meningitis occurs secondary

to a known tuberculous focus elsewhere. This was found to be rare in childhood and compared with primary tuberculous meningitis it represents considerably less, only 7 per cent, of the total diseases of the pia mater. In 60 per cent of the secondary cases the meninges became involved within less than a year after the primary clinical lesion was discovered. Contrary to the general opinion, acute infections did not seem to precipitate a generalized tuberculosis. The seasonal influence was definite, more cases occurring in the spring with a less marked rise in the fall. Other factors such as change of climate or surgical trauma seemed not to have a direct bearing on the development of a secondary tuberculous meningitis.

LESLIE, Evanston.

HOW VIENNA IS MEETING THE PROBLEM OF HEREDITARY SYPHILIS. PARADEISER, Rev. internat. de l'enf. **5**:98, 1928.

The great number of children in the city of Vienna found to be suffering from inherited syphilis has practically made it obligatory to devise some means not only to care for these unfortunates but to prevent, so far as possible, further accessions to their ranks. A special pavilion in the Zentralkinderheim was opened for this purpose in 1924. It was at first suggested that parents be forced to arrange for suitable treatment of infected children and that syphilis should be classified among the notifiable diseases. This plan was fortunately not adopted. Wiser counsel advocated the education of the public, and this has led to gratifying results. Every adult female in Vienna who is not a member of a benevolent society has the right to a small weekly subsidy for four weeks prior to confinement provided she makes application in season and submits to the Wassermann test.

In 1925, 429 patients were cared for; in 1926, 768 and during the first six months of 1927, 411. In the first series there were 14 mothers with positive blood tests, in the second series, 16 and for the third group, 26. The report of the bureau shows that 8 per cent of the infected women were under 20 years of age; 50 per cent were between 20 and 30; 39 per cent were between 30 and 40 and 3 per cent over 40 years. Two thirds were married, 7 per cent were divorced and 27 per cent were unmarried. It has been found that when relief was sought early and diligent treatment instituted the infants were born free from disease. Paradeiser predicts that the growth of this health center should do much to reduce the infant death rate in Vienna.

AMESSE, Denver.

DIAGNOSIS OF TUBERCULOSIS BY CULTURE. W. HAUPTMANN and J. BURTSCHER, Wien. klin. Wchnschr. **41**:84 (Jan. 19) 1928.

The authors describe Hohn's method of making cultures of tubercle bacilli. The suspected material is first treated with 10 to 12 per cent sulphuric acid to destroy other organisms. It is then centrifugalized and the sediment transfered to egg-glycerin slants, which, after protection from evaporation, are incubated at 37 C. Hauptmann and Burtscher present modifications of Hohn's technic. They also advise preliminary treatment of albuminous fluids, e. g., serous exudates, with 10 per cent sulphosalicylic acid in order to produce an artificial sediment.

In a series of 114 suspected specimens, in which smears had proved negative or doubtful, the authors were able to produce a growth of tubercle bacilli by the cultural method in 83 instances. They found the method most valuable for sputum, urine, pleural exudates, tissue from the bones and joints and the stools of infants. It was limited in scope to specimens containing living tubercle bacilli, which were capable of propagation. Positive results could be obtained in a shorter time by culture than by animal inoculation; the authors were able to find the organisms by Hohn's method in from eight to sixty-four days.

The authors conclude that the cultural method described is sufficiently simple to be applicable to routine laboratory diagnosis in cases in which smears have proved negative.

DIFFERENTIAL DIAGNOSIS OF TUBERCULOUS AND NONTUBERCULOUS PULMONARY DISEASE IN CHILDHOOD. HANS WIMBERGER, Wien. klin. Wchnschr. **41**:303 (March 1) 1928.

Wimberger calls attention to the difficulty of differentiating tuberculous from other chronic infections of the lung, particularly in older children having positive tuberculin reactions. He stated that roentgenograms offer strong support to clinical diagnosis, especially when serial plates covering a considerable period of the patient's illness are available.

In early life, a central or peripheral primary focus and the surrounding "perifocal" area of infiltration may be confused with pneumonia. A history of gradual onset or of a tuberculous environment speaks for a tuberculous process. One should always search the skin for tuberculids. The author disagrees with the popularly held notion that tuberculosis does not attack the apexes of children. He asserts that in an individual roentgenogram, one cannot differentiate a tuberculous from a nontuberculous infection of the hilum region.

Wimberger calls attention to certain secondary infiltrative changes which he and other investigators have observed to take place around primary foci of tuberculosis in the lungs, and which he attributes to the effects of specific endotoxins. They consist of minute or gross areas of edema of the parenchyma. They often resemble pneumonic processes but differ from these in the tendency not to remain limited to the boundaries of a single lobe. The edema may be transient; it may recur, or it may become chronic and lead to induration or contracture of the lung. True caseous tuberculosis may follow in certain cases. Large areas of secondary infiltration, on examination, are characterized by diminution of pulmonary resonance without much deviation from the normal auscultatory symptoms.

The author has noted the frequent localization of chronic bronchopneumonia in the lung bases, especially after grip, pertussis and measles. He states that this form is rarely tuberculous. It often ends in the formation of pleural adhesions and bronchiectasis. The character of the tuberculin reaction may aid in diagnosis, a marked reaction indicating tuberculosis.

OLMSTED, Seattle.

SYPHILIS AND INJURY TO THE EMBRYO. J. HUSLER and A. WISKOTT, Ztschr. f. Kinderh. **43**:555 (July) 1927.

The question whether syphilis in the parents can cause injury to the embryo and subsequently an inclination to the disease in future generations and whether this can occur without simultaneous transmission of syphilis is still a disputed one. Husler and Wiskott examined the descendants of parents with congenital syphilis with a view to establish whether injuries attributable to syphilis could be found seldom or frequently, regularly or irregularly. Among the patients seen in the University Clinic of Munich from 1887 to 1906, forty with congenital syphilis who were of the procreative age were found and studied with special attention to their fertility and the condition of their descendants. It was found that the syphilis had remained latent since childhood, although the Wassermann reaction continued positive to a late age. Their fertility was not diminished, and the ratio of full term to premature births or abortions was the same as that of healthy people. The physical and mental make-up of the living descendants, forty-two in all, was similar to those of the average nonsyphilitic persons. Syphilis as such could not be demonstrated in the third generation and even a parakinetic injury to the embryo by syphilis, that is, not a direct injury to the tissues by inflammation but an indirect one by the action of toxins on the endocrine organs affecting development — was by no means evident, even under conditions of long activity and inadequate treatment.

UNILATERAL HYDRARTHRUS WITH BILATERAL GUMMATOUS EPIPHYSITIS OF THE TIBIAE AND PSYCHIC INFANTILISM. TRENDTEL, Ztschr. f. Kinderh. **43**:580 (July) 1927.

Trendtel believes that there are still forms of late syphilis which are worth investigating and describes a syphilitic boy, aged 7 years, with a severe hydrops of the right knee, bilateral osteitis and gummatous epiphysitis in additon to psychic infantilism.

EMERSON, Boston.

Internal Diseases

PYELITIS OF INFANCY AND EARLY CHILDHOOD. THOMAS W. MARKS, Kentucky M. J. **26**:63, 1928.

The author believes that the mode of infection in pyelocystitis is both hematogenous and ascending, more likely being hematogenous in primary cases and ascending in cases following acute gastro-enteritis.

The alkaline treatment is best during the acute stage of high fever and vomiting because it is safest and most effective at this period. As acute symptoms subside the acid-methenamine treatment may be employed, from 20 to 30 grains (1.3 to 1.95 Gm.) of methenamine in twenty-four hours being used. Hexylresorcinol is effective in chronic forms which resist the alkaline and acid-methenamine methods. Since fluids cannot be pushed with hexylresorcinol, it cannot be recommended for acute forms of the disease in which fever is present. Patients whose condition does not clear up on medical treatment after a reasonable time should be referred to a urologist for cystoscopic examination.

BRUCE, Louisville, Ky.

INTRA-ABDOMINAL HEMORRHAGE IN A BOY OF SIX YEARS. T. M. MILLAR, Trans. M.-Chir. Soc., Edinburgh M. J. **35**:21 (Feb.) 1928.

Millar showed a young patient who had had rupture of the liver from trauma, with massive hemorrhage, and who had been cured by laparotomy and gauze packing, without the rent in the liver being sewed up. Immediate blood transfusion of one and one-half pints (710 cc.) was given. The child made a good recovery in three weeks.

TYPES OF BRONCHIECTASIS IN CHILDREN. CHARLES MCNEIL, Trans. M.-Chir. Soc., Edinburgh M. J. **35**:24 (Feb.) 1928.

Two boys were shown, in whom the injection of iodized oil 40 per cent into the bronchial tree illustrated two different types of bronchiectasis. One of the children, aged 4½ years, had a double saccular bronchiectasis near the vertebral column. The other child, aged 9 years, had displacement of the heart to the right nipple; the shadow caused by the iodized oil showed the trachea to the right of the vertebra, the main branches of the bronchus ending in thick, clublike dilatations. The esophagus also was on the right side. In addition to bronchiectasis, the lower lobe of the right lung was contracted and fibroid, with displacement of all the structures of the mediastinum to the right. Judging from reports of adults who had a history of bronchiectasis from early childhood, McNeil believes that ultimately the health of these persons may become good.

PNEUMOTHORAX IN A CHILD OF SIX YEARS. W. A. ALEXANDER, Trans. M.-Chir. Soc., Edinburgh M. J. **35**:33 (Feb.) 1928.

Alexander presented a case of pneumothorax in a girl, which had followed the spontaneous evacuation of pus by the bronchus from the right pleura. The right lung, which had been contracted, had become fully expanded, and the child became well.

NEFF, Kansas City.

THE MAXILLARY FACIAL DYSTROPHIES. J. MEYER and NICOLLE, Rev. franç. de pediat. **3**:304, 1927.

In this group of dystrophies, the authors place those cases which show deformities of the upper and lower maxillae that come on in infancy in the absence of any traumatism and often lead to functional disturbances. In general, the face appears small in proportion to the rest of the head; the jaws are narrow; there may be either prognathism or retrognathism; the palate is highly arched, and the teeth do not meet properly. From these deformities a diminution in the nasopharyngeal and pharyngobuccal spaces results. This may lead to the formation of adenoids or, when retrognathism is present, the base of the tongue may impinge on the epiglottis and partially obstruct the opening into the larynx. In consequence, then, a certain amount of anoxemia, insufficient movement of the diaphragm, and certain circulatory disturbances may result. Thus the individual is more or less handicapped, and there may be a persistence of an infantile type. The authors are inclined to think that the primary disturbance takes place early in intra-uterine life, but the exact etiology is obscure. Syphilis is considered but is probably not a causal factor, although children of this type are more apt to show stigmas of congenital syphilis than other children in the same family. The whole discussion is extremely interesting and the subject is a significant one, not often dealt with in pediatric literature.

A STUDY OF THE FUNCTIONAL CAPACITY OF THE KIDNEYS AND ITS IMPORTANCE IN PRACTICE. MAX. M. LEVY, Rev. franç. de pediat. **3**:772, 1927.

The author emphasizes the importance of the power of the kidney to concentrate urea as a test of renal function. The normal maximum concentrating power is from 50 to 55 Gm. per thousand cubic centimeters. Experimentally this may be determined by giving the subject a diet rich in proteins and poor in water. Clinically, conditions of oliguria offer the opportunity to study the concentrating power of the kidney. The determination of this factor is an aid in prognosis, a rapid decrease pointing to a fatal outcome. In many cases of edema, the concentrating power is normal; also in persons with cardiac disease in whom it might otherwise be difficult to determine the functional capacity of the kidney, this test is of value.

NIEMANN-PICK'S DISEASE. CORCAN, OBERLING and DIENST, Rev. franç. de pediat. **3**:789, 1927.

The article gives a full discussion of Niemann-Pick's diseases with a new case report, the eleventh, and fourteenth plates showing the pathologic histology in the case studied. The case was first treated as one of congenital syphilis, but when results were not obtained from antisyphilitic treatment over a period of three months, biopsy was performed. Microscopic examination showed the lipoidocellular infiltration of Niemann-Pick. The clinical features were cachexia, marked enlargement of the spleen, liver and lymph glands, a brownish yellow pigmentation of the skin and an extreme inertia. The blood showed a cholesterinemia. Study of the material obtained at autopsy showed extensive infiltration with large vacuolated cells with eccentric nuclei containing a mixture of neutral fats and lipoids. Occasionally, the parenchyma itself seemed to have changed into the characteristic type of cell. These cells differ from Gaucher's cells in that in the latter the material in the protoplasm is almost insoluble in fat solvents.

The etiology is obscure, but the early onset and the frequent coexistence of malformations, mongolism, amaurotic familial idiocy and, in the case studied, microgyria, point to some constitutional defect. The disease process is also obscure. The possibilities of the condition being due to an infiltrative process or to a perverted activity of the reticulo-endothelial cells are discussed.

LESLIE, Evanston.

NERVOUS SYMPTOMS PROVOKED BY INTESTINAL WORMS. A. KOLLMANN, Arch. f. Kinderh. **82**:150 (Sept. 30) 1927.

Kollmann reports two cases, one in a child aged 3 years and one in a child aged 4, who showed symptoms of a disturbance of the central nervous system. These were rigidity of the neck, a positive Kernig sign and lively patellar reflexes. The spinal punctures resulted in normal fluids. Oxyuria were found in the stool, and prompt recovery followed their treatment.

RUMINATION IN CHILDHOOD. G. ABRAHAM, Arch. f. Kinderh. **82**:199 (Sept. 30) 1927.

Abraham uses both a dietetic and a psychic form of treatment. He thinks that some form of thick cereal feeding should be introduced as soon as possible even with breast feeding. The actual act of rumination is hampered by placing the child on its abdomen, or tying the jaws together. He reports three cases which illustrate these methods.

SUGAR TREATMENT OF CHILDREN WITH NEPHRITIS. W. HIRSCHFELD, Arch. f. Kinderh. **82**:245 (Nov. 11) 1927.

In the author's series of cases there was no effect on any of the patients in regard to the symptoms of albuminuria or hematuria that could be attributed to the sugar. The sugar treatment did, however, appear to be of value in those cases of nephritis occurring with uremia, or in which uremia was imminent.

POLYARTHRITIS DEFORMANS IN CHILDREN. W. E. JOLKWER, Arch. f. Kinderh. **82**:273 (Nov. 11) 1927.

The author states that arthritis deformans in children is identical with the same condition in adults. Many of these conditions may be caused by a thyroid deficiency. The author calls these cases in which direct evidence of a pathologic condition of the thyroid is present, thyrogenous arthritis. He also believes that in all cases of chronic disease of the joints one should make an extensive study into the functioning of the thyroid.

SANFORD, Chicago.

ACUTE RAPIDLY FATAL OSTEOMYELITIS. FRITZ SCHURER-WALDHEIM, Arch. f. klin. Chir. **144**:65, 1927.

Acute fulminating fatal osteomyelitis presents a characteristic clinical picture. Opinions differ now only in regard to therapy, though the etiology and mechanism of the disease are still subject to some dispute. However, it is beyond dispute that when micro-organisms (pyogenic) enter the blood stream they produce osteomyelitis on an embolic basis; therefore, since the disease affects young persons, it is regarded as a pyemic disease of the growing period of the child. The author regards osteomyelitis, therefore, as always secondary, even in the absence of a history of infection, such as furuncles, etc., for the mucous membrane may be penetrated by bacteria without noticeable damage to the mucosa.

The author lists under this fulminating types such as peri-ostitis albuminosa, osteitis nonpurulenta and hemorrhagic septic forms with early death. He reports six deaths from this fulminating type. All the patients died within from three to six days after the onset, three dying during the first three days, and the author states that he has been unable to find any reports in the literature in which death has occurred so quickly. In his series the tibia and pelvic bones each were involved twice, and the femur and scapula each once. In four there was an associated exudate in the adjacent joint. Four of the six patients were males in whom the infection was preceded by trauma. The author believes that bone marrow is a splendid culture media, since lymphoid tissue is most abundant in the young. The causative organism was *Staphylococcus*

aureus. Briefly, the clinical and physical signs are pain, swelling and fever in the involved bone, followed by chills, high fever, delirium and, sometimes, a rash. The local lesion soon develops into a rapidly spreading abscess which extends out to involve the surrounding structures and produces a so-called phlegmon with infection of the joint and formation of a thrombus. At autopsy, metastatic abscesses invariably are found throughout the body, producing a severe form of septicopyemia.

The treatment is always surgical and to be of value must be instituted early. Therefore, an early diagnosis made before changes occur that show roentgenologically is imperative.

OBERHELMAN, Chicago.

LYMPHATIC REACTION IN GRIP. CORNELIA DE LANGE, Monatschr. f. Kinderh. **37**:202, 1927.

De Lange reports the case of an infant, aged 9 months, who during an attack of ordinary grip showed a white cell count of more than 41,000. Of these, 84 per cent were lymphocytes. The red count was normal. The infant recovered.

A DIABETIC CHILD WHO WAS RESISTANT TO INSULIN. P. SZTAVROVSZKY and K. WALTNER, Monatschr. f. Kinderh. **37**:214, 1927.

In a diabetic child, first one brand of insulin and later another was used with little result. As high as 300 units divided in from two to four doses were given daily without making the child sugar free or producing toxic effects. In this child the red cells fixed 8 per cent less glycogen from the blood serum than normal.

GERSTLEY, Chicago.

ARRHYTHMIAS OF CHILDHOOD. A. F. HECHT, Wien. klin. Wchnschr. **41**:232 (Feb. 16) 1928.

Hecht calls attention to Pirquet's observation that the pulse period, which is the reciprocal of the pulse frequency, is proportional in early life to the sitting height, except in the new-born. Here the pulse frequency is relatively lower on account of the large heart.

Tachycardia and bradycardia are often erroneously diagnosed because physicians fail to consider the normal pulse rate of children at various ages. Hecht mentions Helmreich's demonstration that the minute volume blood flow does not vary during the tachycardia induced by a change from the horizontal to the upright position (static acceleration), but that the volume flow may increase as much as four times during exercise (dynamic acceleration). In infantile tetany, tachycardia occasionally is seen; the attacks begin and end suddenly. Juvenile tachycardia does not lead to auricular overfilling as easily as in adults, because the duration of conduction time and ventricular systole is relatively shorter.

Sinus bradycardia was observed by the author in a boy, aged 12 years, with diabetic coma. He has noted that sinus arrhythmia may become so accentuated at the age of puberty as to lead to palpitation. Digitalis is contraindicated in the arrhythmia of diphtheria, which is due to vagus hyperirritability. Extrasystoles are uncommon before the tenth year and are often diagnosed when they do not exist, as shown by electrocardiographic studies. They are found occasionally in persons with angina, rheumatic endocarditis and diphtheria.

The author observed sisters with paroxysmal tachycardia, which he viewed as the expression of an inferior circulatory apparatus. Auricular flutter is found in diphtheria, scarlet fever and grip and is well borne in childhood. For its treatment Hecht employs quinine (0.2 to 0.3 Gm. daily). He has seen only two children with auricular fibrillation. Both were at the age of puberty and had severe mitral lesions.

Disturbances in conduction, according to the author, are seen in a number of acute infections, and they usually spell myocardial disease. Occasionally, the conduction apparatus alone is involved. Hecht observed Stokes-Adams syndrome in a girl, aged 3 years and 6 months, after an attack of laryngeal diphtheria.

OLMSTED, Seattle.

UROGENITAL DISEASES IN INFANCY: II. THE FREQUENCY OF URINARY SYMPTOMS IN INFANCY. SIEGFRIED LEVY, Ztschr. f. Kinderh. **43**:516 (May) 1927.

The characteristic nephritis of infancy is rare and has an unfavorable outcome. On the other hand, urinary symptoms are frequent which do not, however, influence the prognosis of the primary disease. In a study of 2,800 histories from the Infant's Hospital at Hamburg from 1918 to 1926, there were mild urinary symptoms in 5.9 per cent of all the infants, which signified a functional weakness of the kidney. The mortality of patients with urinary symptoms was about twice as high percentum as the total mortality. Especially significant was the difference which appeared in the consideration of the factor of sex, the mortality and frequency of urinary symptoms being much higher in boys. The occurrence of urinary symptoms was represented in tables showing different diseases and the frequency of urinary symptoms with respect to them. From the presence of these symptoms, a conclusion as to prognosis could not be established, but an impression of the severity of the illness in contrast to that of the nephritis itself could be obtained. Especially noteworthy was the frequent occurrence of albumin, leukocytes, red cells, etc., in tetany, and the rarity of their occurrence in eczema. The different causes which came into the consideration were discussed. On the grounds of these points, the importance is again emphasized of regular examinations of the urine in the clinic as well as in private practice.

A CASE OF FUNCTIONAL PARTIAL HEART BLOCK. ERNST LORENZ, Ztschr. f. Kinderh. **43**:552 (May) 1927.

If one glances over the literature describing cases of heart block in children, one will find chiefly two forms; the congenital, usually total, heart block, which is found frequently with congenital malformations of the heart, and the post-diphtheric form of later childhood, which occurs after organic injury of the conduction system. The bacillus of Klebs-Löffler plays a big rôle in the etiology of heart block in children and causes 30 per cent of all cases. A smaller number of cases of heart block is due to a purely functional disturbance of conduction, and a case of this kind is here discussed.

IS THERE A COLICYSTITIS? (SEROLOGIC STUDY OF THE PYURIA OF INFANTS.) FRIEDRICH STRUNZ, Ztschr. f. Kinderh. **43**:537 (May) 1927.

Authorities differ as to the strain of *Bacillus coli* found in pyuria and their points of entrance into the urinary passages. Whether *B. coli* really plays the primary rôle in the pathogenesis of pyuria or, as saprophytes, overgrows the real and unknown etiologic agent, is disputed. The presence of bacteria without pus seemingly indicates the harmlessness of *B. coli* on the one hand and on the other, as pointed out by Schiff and Bayer, nonbacterial causes may call forth a pyelitis; for example, dehydration. The strains of *B. coli* isolated in pyuria in infants do not form a serologically uniform group and do not point to a special receptor relationship among them. On the contrary, strains of *B. coli* grown simultaneously from the bladder, vagina and the stool of the same patient show a great receptor relationship, and the immune serum obtained from them was similar in regard to serum glutination. This points to a very close relationship, if not to the identity of these strains from the same patient. It is not therefore possible, at least in a serologic sense, to assume the existence of a special colipyuria or colicystitis.

EMERSON, Boston.

THE EFFECT OF INSULIN ON THE DISEASED MYOCARDIUM. I. HAYNAL, Orvosi hetil. **38**:1085, 1927.

Bad effects have been seen from the use of insulin on diabetic patients who had a damaged myocardium. The effects are usually in the form of attacks of angina pectoris, and occasionally they have led to the death of the patient. The author has particularly seen disturbances of rhythm on the heart after the use of insulin and thinks that this may occur in connection with the hypoglycemia produced by the insulin. Determinations of blood sugar and the good effect of injections of glucose support this theory. His conclusions are as follows:

Insulin should be used on diabetic patients with a diseased myocardium only on strict indications. In such cases insulin should be given simultaneously with intravenous injections of glucose. In diabetic coma with cardiovascular involvement, a large amount of insulin should be given with glucose.

ZENTAY, St. Louis.

A PATIENT WITH PAROXYSMAL TACHYCARDIA TREATED WITH APOMORPHINE. E. M. CHRISTENSEN, Ugesk. f. Laeger **90**:186, 1928.

The history is given of a girl, aged 19 years, suffering from congenital pulmonary stenosis and paroxysmal tachycardia. Of several drugs used to relieve the tachycardia, it was found that apomorphine hydrochloride, 1 cc. of a 1 per cent solution subcutaneously, relieved the attack in ten minutes.

SCHLUTZ, Minneapolis.

Nervous Diseases

PINEAL PATHOLOGY. G. HORRAX and P. BAILEY, Arch. Neurol. & Psychiat. **19**: 394 (March) 1928.

The authors feel that the function of the pineal body in the human organism is still a subject of controversy. Three cases are presented which support this view. The first case was in a boy, aged 9. In this instance the pineal body was replaced by a malformation, and there is no evidence that any normal pineal parenchyma ever existed; yet the patient attained normal physical and sexual development. In the second case a typical ganglioneuroma was found formed from the pineal anlage. The third case was that of a boy, aged 7. Necropsy showed the testes to be similar in structure to those of a full grown man. A cyst was present in the pineal body, which was assumed to be a dilatation of the cavum pineal, since it was surrounded by a thick layer of neuroglia. As the tumor was not a teratoma or a pinealoma, the syndrome cannot be attributed to a secretion furnished by the tumor. Neither can it be explained as a destruction of a hypothetical brain center, as numerous tumors that are unaccompanied by precocious puberty are found in this location. There is no explanation, therefore, for the essential cause of the macrogenitosomia praecox of which this case was such an excellent example.

CEREBELLAR TUMORS OCCURRING IN IDENTICAL TWINS. F. H. LEAVITT, Arch. Neurol. & Psychiat. **19**:617 (April) 1928.

Two cases are presented of cerebellar tumors in twins that were apparently of the monozygotic or identical type. The first twin died at the age of 6½ years, and the second died when 8½ years of age. The author believes that the identical occurrence of cerebellar tumors in monozygotic twins may be construed as evidence strongly supporting the "fetal rest" theory of the development of the neoplastic tissue. There can be little doubt that the twins had cerebellar neoplasms in similar situations, and as the structure of the tumor examined was composed of embryonic cells, and, as it is believed that monozygotic twins develop as a result of the splitting of a single fertilized ovum, it would appear that both of these tumors were due to a "fetal rest"

of embryonic tissue. This tissue remained as a pathologic nidus in the roof of the fourth ventricle of each twin in a quiescent state from the time of the splitting of the original single ovum until several years later when some factor stimulated the pathologic growth of this group of embryonic cells in each twin.

HAS THE PEDIATRICIAN ANY PLACE IN THE MENTAL HYGIENE MOVEMENT? B. CROTHERS, Arch. Neurol. & Psychiat. **19**:676 (April) 1928.

From the pediatric point of view, an interest in mental processes is obviously all that keeps the specialty within the field of general practice; therefore, pediatricians should cooperate with psychiatrists. Psychiatrists might well make serious efforts to fit pediatricians into a scheme of mental hygiene. The issue seems to be clear. If mental hygiene is a separate branch of science for which technical tools are of a predominant value, few pediatricians will enter it; if it is one of the most vital parts of the practice of medicine, it will appeal to the best pediatricians as a new, vividly interesting task to be entered on with fear but also with enthusiasm. Psychiatry as a dignified specialty does not need any defense from scoffers, but it does need protection from those who suggest that it can be divorced from medicine and placed in the hands of technicians.

SANFORD, Chicago.

STAPHYLOCOCCUS INFECTION OF SPINAL MENINGES. KENDALL EMERSON, Boston M. & S. J. **196**:465 (March 24) 1927.

Emerson reports an interesting case of apparently localized infection of the spinal cord in which a generalized spinal meningitis did not develop.

A girl, aged 12 years, was taken ill in school a week before admission to the hospital with severe pain in the left hip. An irregular temperature ranged up to 103 degrees; nausea and vomiting occurred three days later, the nausea persisting. The day before admission the child had had some pain in the back and marked constipation, and the next morning increasing headache, slight rigidity of the neck and spasticity of the legs were present. The previous history revealed nothing of importance. The ears, eyes, nose, throat, heart and lungs were normal. The lower abdominal reflex was absent; there were moderate loss of pain and tactile sense in both legs and marked diminution in pain and tactile sense in the saddle-back area involving the third and fourth sacral segments. Tache cérébrale and double Babinski signs were present, but the Oppenheim and Gordon signs were absent. Lumbar puncture with a no. 18 needle between the second and third lumbar vertebrae gave negative results even with suction. Cisternal puncture gave clear fluid under slightly increased pressure which proved sterile. Lumbar puncture performed the next day with a much larger bore needle again gave negative results, but with heavy suction 2 cc. of thick whitish pus was obtained. Before operation two days later, cisternal puncture gave clear fluid, and lumbar puncture from the first lumbar segment gave blood tinged fluid, and from the second and third lumbar spaces, thick pus. Laminectomy of the second and third lumbar vertebrae was done, and when the canal was opened the dura was found to be greatly injected and bulging. On incision, bloody purulent fluid escaped, which showed *Staphylococcus aureus* and gram-negative bacilli on culture. Cerebrospinal symptoms began to clear up within twenty-four hours. Tube drainage was used for about ten days. The child was discharged fifty days after the operation; the sinus was closed; the spine showed normal flexibility, and roentgenograms did not show any evidence of osteomyelitis of the vertebra. Two months later the child developed osteomyelitis of the right elbow joint; this was followed in several months by involvement of the right radius. The child remained well for a year, and then had an abscess in the right groin from involvement of the ilium. The spinal region continued to be free from infection, as shown by subsequent roentgen-ray examinations.

GENGENBACH, Denver.

CEREBROSPINAL FLUID IN CHILDREN. M. G. PETERMAN, Wisconsin M. J. **27**:59 (Feb.) 1928.

Two hundred spinal fluids obtained from infants and children were examined in detail. All the familiar tests for spinal fluid were carried out, and in addition the microprecipitation test for syphilis and the Levinson protein precipitation test were made. Cerebrospinal fluid was taken from forty-four patients who were not ill. The cell count ranged from 0 to 6 cells, (lymphocytes), the average being 1.8 cells; sugar ranged from 32 to 117 mg. per hundred cubic centimeters; the Levinson test was negative.

In seven cases of congenital syphilis, in which Wassermann and microprecipitation tests on the blood were four plus, the Wassermann and microprecipitation tests on the spinal fluid were positive in only two. The Levinson test was characteristic in thirty-two fluids from seventeen patients with tuberculous meningitis. Tubercle bacilli were demonstrated in smear of the pellicle in fourteen cases. (In infectious meningitis, the simplest tests are smear and culture for organisms.)

Spinal fluid should be obtained from every patient with unexplained fever and signs of meningeal irritation. A complete examination of the fluid made immediately will often establish a diagnosis. The Levinson test is a valuable one in tuberculous meningitis.

The author warns of the danger of cisternal and ventricular puncture in unskilled hands.

KELLY, Milwaukee.

THE APPLICATION OF CHROMONEUROSCOPY IN CHILDREN'S CLINIC ACCORDING TO THE METHOD OF FLATEAU. V. MIKULOWSKI, Rev. franç. de Pédiat. **3**:845, 1927.

Normally, in man, acid fuchsin injected intravenously or intramuscularly does not appear in the cerebrospinal fluid, but in pathologic conditions, notably in meningitis, it may be recovered from the cerebrospinal fluid within from one and one half to two hours. The reagent used for the detection of the acid fuchsin consists of one part hydrochloric acid and nine parts 95 per cent alcohol. The spinal fluid is collected in a small porcelain cup which contains a few drops of the reagent; a violet color appears in the fluids giving a positive reaction. The test was used in seventy-three cases. In fifty-four it was positive; these cases consisted of five of epidemic meningitis, five of pneumococcic meningitis, three of serous meningitis and forty-three of tuberculous meningitis. The nineteen cases showing a negative reaction had to do with cerebral congestions, meningismus occurring in the course of pneumonias and typhoids, nephritides, alimentary intoxications and, in one instance, cerebellar tumor.

LESLIE, Evanston.

A RARE SEQUELA OF EPIDEMIC ENCEPHALITIS. A CONDITION SIMULATING MULTIPLE SCLEROSIS OF THE BRAIN AND SPINAL CORD IN AN INFANT. ALBERTO ROSTAN, Riv. di clin. pediat. **25**:697, 1927.

At the age of 2 months, the patient had a severe illness, characterized by lethargy, vomiting, ptosis of the eyelids, paralysis of the ocular muscles, weakness and irregular fever; a diagnosis of epidemic encephalitis was made. When the child was 5 years of age, examination showed: ataxia, mental retardation, peculiarity of character, slow, scanning speech, exophthalmos, nystagmus, weakness of the internal rectus muscle, slow reaction of the pupils to accommodation, deviation of the tongue to the right, intention tremor, positive Romberg and Babinski-Oppenheim signs, diminution of some tendon reflexes, absence of abdominal reflexes, unequal cremasteric reflexes, alteration of thermic sensibility and loss of sense of position.

After discussion of the differential diagnosis, the author concludes that the condition so characteristic of disseminated sclerosis of the brain and spinal cord is a sequela of epidemic encephalitis.

OPPENHEIM'S DISEASE. A CLINICAL AND HISTOLOGIC CONTRIBUTION. IVO TOMASSINI, Riv. di. clin. pediat. **25**:714, 1927.

A biopsy was performed and a specimen of the gastrocnemius muscle obtained from a boy, aged 3 years, with Oppenheim's disease. Marked dystrophy of the tissue with proliferation of the nuclei was found; histologic sections are illustrated in the article.

The differential diagnosis of amyotonia congenita and Werdnig-Hoffman's type of progressive muscular atrophy is discussed. The former is congenital but not familial; the muscles do not show evident gross atrophy, and the patient tends to improve. The latter is usually found in families and becomes evident some time after birth; atrophy of the muscles is grossly apparent, and the patient tends progressively to get worse.

HIGGINS, Boston.

THE TREATMENT OF PURULENT MENINGITIS FROM THE PEDIATRICIAN'S STANDPOINT. K. STOLTE, Beitr. z. klin. Chir. **139**:176, 1927.

The author discusses briefly the symptoms, diagnosis and treatment of purulent meningitis. He cautions that in lumbar punctures one should not forget that the cerebrospinal fluid may be purulent in tuberculous meningitis and that a serous fluid low in albumin and cell count may be the beginning of an acute infectious meningitis.

The organisms most commonly found in purulent meningitis are the streptococci, staphylococci, pneumococci and meningococci, although the bacillus of influenza, dysentery, typhoid and paratyphoid may be responsible. With such a variety of bacteria responsible for a purulent meningitis, one must know what organism is present to render a prognosis.

The treatment of purulent meningitis is conducted along symptomatic and etiologic lines. The symptomatic treatment consists of the application of ice packs, the administration of anodynes, infusions of fluids and repeated lumbar puncture to relieve the increased intracranial tension. The treatment along etiologic lines consists first in determining the source of the infection, and if the source is local, such as an otitis media, operative measures should be employed for establishing drainage. The author states that a purulent meningitis formerly had lent itself to only a 3 per cent recovery, but now that operative drainage has been instituted, the recovery has been increased to at least 30 per cent. However, if the meningitis is hematogenous, it is then only a part of a general infection. Here the author proposes two methods of treatment of which one or both may be used. The one is the drug and the other the serum treatment, both of which must be suited to the type of causative organism. In the absence of specific serum, good results have been obtained by the author from massive doses of methenamine (from 6 to 10 Gm. daily), and also from arsphenamine in syphilitic meningitis, optochin in pneumococcic meningitis, and a flavine dye in streptococcic and staphylococcic meningitis. Optochin, though more or less specific in pneumococcic meningitis, has been found useful in meningococcic meningitis. In case of abscesses of the brain, the author has used the flavine dye, also encupin and vuzin with good results.

With regard to serum therapy the author is enthusiastic, and for the administration of such he urges early diagnosis of the condition and causative organism. He favors the use of massive doses, given intravenously or intramuscularly in preference to intraspinally, because, in case of purulent meningitis, the brain and spinal cord are covered with fibrin and prevent any of the serum from penetrating the tissues in which the serum is most needed; he believes that if the serum is given intravenously or intramuscularly, the blood will carry it into the tissues. Dosages as large as 150 or 200 cc. are given daily for three days, and in one instance the author gave up to 340 cc. in two days. The author proved this to his own satisfaction in two cases of purulent meningitis due to the streptococcus organism in children, and both made splendid recoveries from such massive doses.

The author's conclusions, therefore, are early drainage in purulent meningitis which has developed as a direct extension from a nearby focus, and massive doses of proper serum given intravenously or intramuscularly for a hematogenous purulent meningitis.

OBERHELMAN, Chicago.

URANIN AS AN AID TO THE DIAGNOSIS OF MENINGITIS IN CHILDHOOD. CARLA FEIGEL, Ztschr. f. Kinderh. **43**:585 (July) 1927.

Jervell's test for meningitis, the action of uranin on the spinal fluid, has the advantage of simplicity. This color substance in doses of 0.03 Gm. per kilogram by mouth for children and 0.02 Gm. for smaller children and infants is nonpoisonous and does not enter the normal cerebrospinal fluid but in all patients with meningitis can be demonstrated with great ease. The drug is given to children in marmalade and to infants in milk and two and one-half hours later, from 3 to 4 cc. of spinal fluid is examined for fluorescence. Eighty children and infants were tested; nineteen of them had various forms of meningeal inflammation, and in all who had meningitis there was an outspoken fluorescence of the fluid. Such a far reaching parallelism exists between the outcome of this reaction and that of Pandi that it seems permissible to conclude that the Pandi reaction is essentially a test of the permeability of the meninges for albumin. Uranin is a still finer medium, but its value lies clinically in its clear visibility. There are times when the Pandi test is positive, in patients in whom meningitis does not exist. When the Pandi test is positive but the test with uranin is negative, one may doubt the diagnosis of meningitis, while the diagnosis is never in doubt when both tests are positive.

THE THYROTOXIC SYMPTOM-COMPLEX IN CHOREA MINOR. K. KUNDRATITZ, Ztschr. f. Kinderh. **43**:658 (July) 1927.

On the ground of observations and experience with fifty-four patients with chorea minor, as well as comparison of this condition with the symptoms of hyperthyrosis, the author believes that he is justified in assuming that a number of symptoms in the fully developed picture of chorea are traceable to thryotoxic influence: the widely open eyes, their brightness, the lag of the lids often in contrast to the general unrest, the easy flushing, the expression of apprehension and anxiety, the sudden breaks in the voice and disturbances of breathing. Not all of the cardiac anomalies observed in chorea can be referred to rheumatic infection and may be thyrotoxic in origin, e. g., palpitation. Other similarities in the two diseases that are present, but not always constant, are enlargement of the thyroid, lymphocytosis, eosinophilia, the reaction to pilocarpine, paresthesia, tendency to glycosuria, increase in basal metabolic rate, loss of weight, excessive incidence and response to infections and the incidence among young females. He does not, however, want to designate these appearances as a complication of exophthalmic goiter but is of the opinion that they belong to the symptomatology of chorea itself.

THE INFLUENCE OF THE KETOGENIC DIET ON EPILEPSY. E. NOBEL and R. WAGNER, Ztschr. f. Kinderh. **43**:748 (July) 1927.

On the basis of their observations on ten children of different ages with epilepsy, the authors could not confirm the observations of Petermann as to the curative effect of a ketogenic diet on epilepsy.

EMERSON, Boston.

A CASE OF APLASIA OF SEVERAL NUCLEI OF THE NERVES OF THE BRAIN. A. H. VAN GELDER and S. WEYL, Nederl. Tijdschr. v. Geneesk. **71**:2344, 1927.

This disease was first described in 1892 by Moebius. It is characterized by the occurrence of nuclear paralyses in the region of one or more motor cranial nerves. The paralyses are congenital or develop during the first year of life. The disease is often familial and is often accompanied by other deviations of

bodily development. Several types of the disease have been described by Moebius and afterward also by other investigators. The first pathologic-anatomic investigation of a case was made in 1902 by Heubner. He concluded that in this disease the nervous tissue in question was built up insufficiently. This opinion was defended later by several investigators.

Here the authors describe a case in a man, aged 53, who had complained of his heart and respiration for about five years. He had never had a serious disease, but since birth he had had difficulty in drinking. Actions and speech had always been faulty since his youth, and he had always been quiet and silent, and had never laughed. The neurologic investigation showed that the patient had a discontented face; it was impossible for him to laugh. The functions of several cranial nerves were found to be totally absent or defective. The hands were asymmetric, and syndactylia was present between the second and third fingers.

S. VAN CREVELD, Amsterdam.

Diseases of the Eye

A CASE OF MALARIAL AMAUROSIS. M. A. NIHAMKINA, J. Pediat. (Russian) **2**:269, 1927.

Considering that malaria in children in the last few years shows severe complications, the author decided to describe a case of malarial amaurosis in a child, aged 1 year and 4 months.

On Nov. 15, 1926, a boy, aged 1 year and 4 months, complained of almost complete blindness of both eyes which had developed twenty-three days after the beginning of a malarial attack. The day of the admission to the clinic, the child was afraid to stand on the floor and wanted to be held by his mother, whom he recognized only by her voice. "If we extended our hands at the same time as the mother, he would touch our hands and become nervous until he would hear his mother's voice." On the third day after his entrance to the clinic, an eye specialist examined the child, and found an inflammation of the optic nerves. After three injections of quinine solution (bichloride, 30 per cent) the child could stand on the floor without fear and without any help. He could even take a few steps in the ward and could recognize his mother without hearing her voice. After the fifth injection, the child was able to take various things, but had to look at them carefully. After the seventh injection, an eye specialist examined the eyes again — the blood vessels were much smaller, the hemorrhage diminished and the boundaries of the optic nerves were definite. After the tenth injection, the child could run about the ward, even in the dark. The nervousness and fear disappeared, and the child became sociable.

The etiology of amaurosis, as a toxic effect of chemicals, could not be applicable in this case, because the patient did not receive any quinine before coming to the clinic. The Wassermann reaction was negative, thus excluding syphilis. Tumor of the brain and meningitis were excluded by a neuropathologist's examination. The kidneys were normal. The child had not had any infectious diseases previously which could have produced amaurosis. It is natural to conclude, therefore, that it was caused in this case by malaria, which is confirmed by the beneficial results of the specific treatment with quinine.

It is notable that amaurosis in this case developed quickly (twenty-three days from the beginning of the first attack of malaria), that sight was restored comparatively quickly, and that the brain was not affected.

SEVERAC, Philadelphia.

Diseases of the Ear, Nose and Throat

A CASE OF BILATERAL DEAFNESS FOLLOWING POLIOMYELITIS. G. BUSACCA and I. TOMASSINI, Riv. di clin. pediat. **25**:780, 1927.

The patient, when 13 months old, had poliomyelitis. Both arms and legs were involved, but three years later the arms alone showed residual paralysis.

Three weeks after the attack, the parents noticed first that the child did not hear common noises. Examination of the child when it was 4 years old showed both labyrinthine and auditory involvement. Cause for the deafness other than poliomyelitis could not be found.

HIGGINS, Boston.

BILATERAL SYMMETRICAL BREAKING DOWN OF THE TEMPORAL BONE, OF THE MASTOID PROCESS AND ALMOST OF THE WHOLE PYRAMID. GEORG JUNG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:211 (Oct. 28) 1927.

In a boy, aged 1½ years, the absence of the temporal bone was noticed during an operation. The mastoid process and the temporal bone were to a great extent replaced by granulations. The defect was almost symmetrical on the two sides. There was a suggestion of aplasia of the temporal bone. Histologic observations, however, showed a bilateral breaking down of both temporal bones, of the mastoid process and of the pyramid. This breaking down was so extensive and occurred so quickly that a specific process was suggested. This could not be proved, however, by any methods at disposal. Therefore, the question remained unsolved.

AMBERG, Detroit.

PULSATION AND VISIBILITY OF THE BULB OF THE JUGULAR VEIN THROUGH THE INTACT DRUM-MEMBRANE, AND REPORT OF SOME CASES. M. SLOBODNICK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:240 (Oct. 28) 1927.

1. The possibility of injury to the bulb of the jugular vein must always be kept in mind in surgical intervention, on account of the relatively great frequency of the protrusion of the bulb of the jugular vein through the floor of the tympanic cavity. This is especially the case in persons suffering from rickets and in children up to 10 years of age.

2. The pulsation of the bulb is dependent on respiratory movements. The muscle system of the neck participates only to a small degree.

3. In two of the three cases reported, the bulb of the jugular vein appeared to be larger when the patients were lying down. This was a mechanical consequence of the greater flow of blood to the brain, and of the smaller outflow in the horizontal position. The aggravation of subjective noises, while the patient was lying down, was attributable in part to the same cause.

AMBERG, Detroit.

Diseases of the Skin

STUDIES OF PIGMENTARY NEVI OF THE HUMAN SKIN (BENIGN MELANOBLASTOMATA). D. A. KISSMEYER, edited by A. LEGRAND.

The author, who spent some time in the year 1918 with Bruno Bloch in Zurich and who became initiated in his methods of studies of pigment by means of the "dopa reaction," has utilized this method in the Rigshospital in Copenhagen in his investigations of pigmentary nevi. The results of these investigations are given in this booklet consisting of 150 pages. After chronologically reviewing the literature on nevi in general, and especially on pigmentary nevi, and after acquainting the reader with the different theories regarding the origin of the "nevus cell," Kissmeyer illustrates how, by means of the dopa reaction, this question can be settled in favor of the epithelia theory first presented by Unna. As the presence of pigment is an important characteristic of the "nevus cell," his investigations on pigmentary nevi coincide with his research concerning the origin of pigment. By many sections stained by both the usual staining fluids and dopa (3, 4-dioxyphenylalanine), he proves convincingly that the nevus cells are originating from the pigment-forming epidermis cells, which give up their connection with the neighboring epidermis cells and are slowly eliminated from the epidermis into the corium. His

investigations show that "lentigo," the flat, not infiltrated pigmented spot, which is so frequently present, forms the first stage of nevus formation, the second stage is the "soft pigmented infiltrated nevus" (the elimination of the nevus cell group from the epidermis has been further progressed). The third stage forms the "papillomatous pigmentary nevus."

By his investigations concerning the origin of pigment, the author confirms the observations of Bloch that pigment is usually formed in cells of ectodermal origin, from which the nevus cell takes its origin. The cells of mesodermal origin, which are found forming the "mongolic spots" and "blue nevi" and which give also a positive dopa reaction, are exceptions.

DAVID BLOOM, New York.

MONGOLIAN BLUE PATCHES. J. D. ROLLUTON, Proc. Roy. Soc. Med. **21**:570 (Feb.) 1928.

Rolluton reported the case of a girl, aged 2 years, the daughter of a Chinaman and an English woman, whose back showed five irregularly shaped areas of slaty-gray pigmentation. These mongolian blue spots are rare in Europeans. They usually disappear when the patient is 7 or 8 years of age.

WILLIAMSON, New Orleans.

RETICULATED AND DIFFUSE PIGMENTATION DATING FROM CHILDHOOD. LAIGNEL-LAVASTINE and SÉZARY, Bull. Soc. franç. de dermat. et syph. **34**:863, 1927.

The patient, a girl, aged 20, had noted a brownish pigmentation of the skin since the age of 7. It was most marked on the abdomen. In some regions it was reticulated; in others it occurred in large patches. There was a slight hypothyroidism, but the authors reserve their opinion about its influence on the genesis of the pigmentation. A biopsy was unobtainable.

APLASIA MONILIFORMIS. MARIO ARTOM, Gior. ital. di dermat. e sifil. **68**:1547 (Dec.) 1927.

The patient was a child, aged 9, who had presented hair dystrophy since the fifth month of life. The father and grandfather also had the same condition. None of the female relatives was affected. The hair dystrophy was accompanied by a marked keratosis pilaris of the neck and limbs. According to the author, the condition is due to a hereditary endocrinous disturbance. Capillaroscopic examination did not show alteration of the circulation of the scalp. The administration of thyroid gland produced some improvement at first, but later the condition remained stationary. Depilation with thallium acetate also failed, the hair showing the same dystrophy after its regrowth.

THE ALKALINE RESERVE OF THE BLOOD IN CERTAIN DERMATOSIS. U. CERCHIAI, Gior. ital. di dermat. e sifil. **68**:1570 (Dec.) 1927.

Investigating the acid-basic equilibrium in the blood, the author finds that in most cases of eczema and psoriasis there is a slight acidosis.

PARDO-CASTELLO, Havana.

PELLAGRA IN CHILDHOOD. BRUNO TRAMBUSTI, Riv. di clin. pediat. **25**:809, 1927.

The author gives a historical and clinical review of the disease.

HIGGINS, Boston.

Surgery and Orthopedics

THE TREATMENT OF BURNS WITH SPECIAL REFERENCE TO THE USE OF TANNIC ACID. S. J. SEEGER, Wisconsin M. J. **27**:1, 1928.

The Davidson method of treating burns was carried out on more than thirty patients at the Milwaukee Children's Hospital. Two and five-tenths per cent

tannic acid was applied to the burns by spraying frequently until the skin becomes brown or black. The burned area is then exposed to dry heat. Dressings are not necessary.

The advantages of this method are:

1. A coagulum is rapidly formed (sixteen hours) over the burned area which prevents, to a large extent, the absorption of toxic substances.

2. The tannic acid solution has an analgesic effect.

3. Healing is hastened because of the protection given by the coagulum.

4. This method of treatment is simple and especially useful in treating children.

KELLY, Milwaukee.

FIBROCYSTIC DISEASE OF HUMERUS. ELIZABETH O'FLYNN and ERIC CROOK, Proc. Roy. Soc. Med. **21**:563 (Feb.) 1928.

This patient was shown six months after a spontaneous fracture. At the operation soon afterward, the upper third of the shaft of the humerus was resected. The pathologic specimen and microscopic sections show the nature of the condition.

WILLIAMSON, New Orleans.

OSTEOSARCOMA OF THE CHEST WALL IN A GIRL OF ELEVEN YEARS. WALTER MERCER, Trans. M.-Chir. Soc., Edinburgh M. J. **35**:37 (Feb.) 1928.

The diagnosis of a roentgenographic shadow in the wall of the chest of this child, in the region of the fourth, fifth and sixth ribs in the left mid-axillary line, lay between a neoplasm and tuberculosis. Thoracotomy at the seventh rib revealed, when the hand was inserted into the pleural cavity, a tumor the size of the child's fist. The growth proved to be an osteosarcoma and was removed by plastic exsection. The result was some collapse of the wall of the chest, but an expanded, well functioning lung. Mercer wonders whether radium should be employed as a prevention of further development.

COMPLICATED FRACTURE OF THE FOREARM IN CHILDREN. D. STEWART MIDDLETON, Trans. M.-Chir. Soc., Edinburgh M. J. **35**:39 (Feb.) 1928.

Middleton showed two uncommon cases which illustrated complications that arose after fracture of the forearm. One girl, aged 6 years, developed a paralysis of the small muscles of the hand and forearm supplied by the ulnar nerve; the dorsal interosseous nerve became involved from a fracture of the upper part of the ulna into the olecranon process, and there was also a dislocation of the head of the radius. If the paralysis, which is disappearing, should not entirely clear up, a surgical exposure of the nerve and a reduction of the head of the radius may be necessary.

Another girl, aged 14 years, following a fracture of both bones of the forearm in the upper third, developed "main en griffe" deformity from the ulnar paralysis, due to inclusion of the ulnar nerve in the callus formation. The paralysis, which is complete, does not show any tendency to recover and operation at the site of the callus will be necessary.

NEFF, Kansas City.

OPERATIONS ON THE SACRAL SYMPATHETIC NERVE IN INFANTS. SOUZA, Arch. de méd. des enf. **31**:86, 1928.

The first successful operation involving the sympathetic nervous system was performed by the English surgeon, Alexander, on Oct. 12, 1883, when he resected the superior cervical ganglion for epilepsy. Twelve years later, Jaboulay popularized operations on the sympathetic in the treatment of persons with exophthalmic goiter; following this, Abadie proposed similar intervention for the relief of glaucoma, although Jannelco was the first to practice it. Jaboulay has also operated on the sacral plexus for pelvic neuralgia, for sciatica and for various trophic disturbances due to gynecologic disorders;

Ruggi has extirpated the utero-ovarian vascular plexus for adnexal neuralgia and Cavazzanni for hysteria. After falling into some disrepute, operation on the sympathetic nerve was revived in military hospitals during the late war and placed on a firmer basis, largely through the work of Leriche, who developed the technic now used in periarterial procedures. Souza reports two cases, the first in a child, aged 2 years, suffering from Raynaud's disease, and the second in a child, aged 8 years, presenting the classic picture of Weir Mitchell's syndrome, that were treated by sacral diuresis. In both of these the results were entirely satisfactory

AMESSE, Denver.

ETIOLOGY OF MADELUNG'S DISEASE OF THE HAND. H. TOLLAS, Arch. f. Kinderh. **82**:112 (Sept. 30) 1927.

Madelung's deformity is a condition of puberty and is rarely ever found in childhood. It should be thought of as of the same process as that which forms a scoliosis, genu valgus or varus, or coxa vara. It is hard to distinguish from the crooked curve produced by an injury. The peculiar configuration of the deformity may be explained as mechanical effort to correct the dynamics caused by the softening bones. The treatment is either conservative by gentle pressure appliances, as advocated by Madelung, or an operative procedure, as advocated by Springer or Gocht.

THE APPENDIX OF THE CHILD IN THE ROENTGENOGRAM. J. SIEGL, Arch. f. Kinderh. **82**:262 (Nov. 11) 1928.

Siegl does not believe that the roentgenologic appearance of an appendix filled with barium is always pathognomonic of disease of the appendix in children. He finds that the filling of the appendix with barium may occur not only when it is inflamed or diseased, but when surrounding structures are similarly affected.

SANFORD, Chicago.

Miscellaneous

DIRECTORY FOR WETNURSES. FRITZ B. TALBOT, Boston M. & S. J. **196**:653 (April 21) 1927.

The establishment of this directory dates back to 1910. The original purpose was to house wetnurses and their children in order to provide a constant supply of human milk for sick infants. The plan was successful but because of the high overhead expense the house was given up, and the milk kept in sterile bottles, collected daily and delivered from a central office. Mothers who produce it are married women with negative Wassermann reactions, found to be in good health by physical examination, and having healthy infants and clean homes. Thirty-five mothers were supplying nearly 500 ounces of milk daily. Tampering is checked periodically by testing the chloride content, which is constant in breast milk; a too low chloride content means dilution with water and a too high content, substitution of some cow's milk. Only one case of tampering was found. Bacterial counts are also taken at intervals. Before being bottled for distribution the milk is strained, pooled, then boiled for one minute, allowed to cool, then restrained and put in sterile bottles. Two thirds of the milk went to private patients who paid 25 cents an ounce, the other third to hospitals at a cost of 12 cents an ounce. Though the value of human milk, especially to sick infants, is fully recognized, many lives have been saved in Boston by the work of the directory.

GEGENBACH, Denver.

A STUDY OF SOME ORGANIC ARSENICAL COMPOUNDS WITH A VIEW TO THEIR USE IN CERTAIN STREPTOCOCCAL INFECTIONS. L. COLEBROOK, Bull. 119, Medical Research Council, 1928.

A small group of trivalent arsenicals comprising neoarsphenamine and sulpharsphenamine, when injected into the animal or human body, confer on

the blood fluids certain bactericidal qualities. In common with other antiseptic agents these arsenicals are capable of destructive action on human leukocytes, given a sufficient concentration and a long enough period of contact, but there is this important difference: whereas the ordinary antiseptics have a higher affinity for the leukocytes than for the microbes and, by combining with the former forfeit their microbicidal potential, these arsenicals have a higher affinity for certain microbes (the hemolytic streptococci and pneumococci) than for the leukocytes. It becomes possible, therefore, by suitable adjustment of dosage, to maintain such a concentration of these arsenicals in a patient's blood as to avoid injury to the leukocytes while conferring a measure of bactericidal potency on the blood fluids.

SANFORD, Chicago.

INFANT WELFARE AND PROTECTION OF MOTHERHOOD IN INDIA. CHESSER, Rev. internat. de l'enf. **5**:67, 1928.

Miss Chesser sees a ray of light in the pall of gloom surrounding child life in India. From a careful study of conditions in Bombay, Puna, Simla, Delhi and many other districts, both urban and rural, she feels that at least an impression has been made against the inconceivable stupidity and incredible ignorance fostered for ages by the changeless superstitions of the native element. Apart from an almost negligible minority, the millions of Indian women are too illiterate to grasp anything more than the bare rudiments of infant and maternal care, but through the influence of clinics established here and there by English women, working under the direction of such organizations as the Lady Reading Hospital of Simla and the Lady Hardinge Medical School of Delhi, many of the dhais, or native midwives, are receiving at least elementary instruction in the conduct of labor. A further advance has been made in the inauguration of the family ward system in the various native hospitals, permitting the young mother to have, if desired, all the members of her family at the hospital during parturition! The dhais are provided with a complete obstetric equipment for their private cases and are encouraged to send for a physician when difficult labor is encountered. In the large towns they are organized under municipal authority and are given half a rupee for each birth reported. The imperative need of multiplying these centers may be imagined from the infant mortality which amounts in many sections to 700 or 800 per thousand.

AMESSE, Denver.

KRETSCHMER'S CONSTITUTIONAL TYPES IN CHILDREN OF SCHOOL AGE. W. S. KRAUSKY, Arch. f. Kinderh. **82**:22 (Aug. 5) 1927.

These studies were made on 100 children of school age with suitable controls. It was found that Kretchmer's teaching of gradation of the body structure with a certain character type held true. It was found that 79 per cent of the children corresponded to this classification, while 21 per cent did not. The principal number of children that deviated from this series of types was found in girls. In girls the gradation of body structure does not correspond to character type as clearly as in boys. The author is enthusiastic about these deviations from the normal types. He believes that a child's classification into one of these constitutional types will be of great aid to the teacher or physician in placing the child in his proper environment. A deviation of the child from his proper type would greatly lighten the work of the teacher or physician and greatly lighten the work of the child, when it is understood that the child is not able to perform the work that is normal for his companions.

THE EFFECT OF PHYSICAL EXERCISE ON THE DEVELOPMENT OF CHILDREN. E. SCHLESINGER, Arch. f. Kinderh. **82**:39 (Aug. 5) 1927.

A study covering several years was made on children and young adults taking exercises in gymnasiums and in graded schools. It was found that not

only were the body stance, heart, pulse and blood pressure improved, but that the entire coordination of the body was greatly increased. A single physical deficiency may be corrected by appropriate forms of exercise, and not only will this one physical defect be improved but all other functions of the body will be greatly benefited. With appropriate sports and graded gymnastic exercises, there will come a facilitation of the coordination of the muscle sense and activity that will greatly improve the dexterity.

SANFORD, Chicago.

INFANT GYMNASTICS AND MASSAGE IN ANCIENT ROME. JENNY AARAU, Jahrb. f. Kinderh. **118**:238, 1927.

Sorannus Ephesios, the famous gynecologist and pediatrician at Rome in the time of Hadrian and Trajan, speaks of massage and gymnastics for infants. A translation is made of the Greek text of Sorannus Ephesios, Book II, Chapter XVI, "About the Bathing and Massage of Infants," which, in brief, is as follows:

Baths should not be long continued and of the proper temperature, to avoid reactions that make the child sensitive to temperature changes, etc. The infant should be bathed once daily, never at night, in a warm place in the direct sunlight. The child should be anointed with oil, and then water at a temperature agreeable to the child allowed to flow over it till the body becomes rosy and warm. The flexures are then cleansed and the mouth also, using the finger dipped in oil. The parts are gently stroked and the lower part of the abdomen also to encourage urination. After the bath the child is to be grasped by the legs and suspended head downward, whereupon it is briskly dried with a linen cloth.

Instructions are next given for moving the arms and legs across the back to hyperabduct them. The limbs are next massaged in their long axis, the bones and joints gently pressed together. The joints are then bent, and massage given in a rotary fashion. The back is then hollowed by massaging the spine from the gluteal fold to the nape of the neck with the middle finger, using pressure. Then the lumbar region is hollowed by pressure with the index and the middle finger and with the closed hand pressure is made over the vertebrae to avoid a lordosis.

The head should be massaged round about and gently molded with the hands into the desired shape. The head is raised, moved and stretched to make the joints and ligaments supple.

The child is then turned on the back and the ventral surface oiled. The arms are massaged downward from the shoulders, and passively moved; thereafter the chest, abdomen and lower legs, the legs being molded by pressure to give them form. With both thumbs the nose is molded into the desired form.

If the foreskin is short, it should be gently stretched till it is of the desired length.

After this the nurse is to anoint the child with oil and wrap it up. Too copious inunction is undesirable. Sometimes it is desirable to anoint the body with Tyrrhenian wax instead of oil, for it keeps the body warm and nourished and whitens the skin.

TRAUTMAN, Milwaukee.

Society Transactions

CHICAGO PEDIATRIC SOCIETY

Regular Meeting, Jan. 17, 1928

MAURICE BLATT, M.D., *President, in the Chair*

NEUROLOGIC DIAGNOSIS IN CHILDREN. DR. LEWIS J. POLLOCK.

The diagnosis of organic diseases of the nervous system in children as well as in adults depends on a knowledge of the antomy and physiology of the nervous system. This knowledge does not have to be intensive, and if a few of the important tracts and their relation to each other as well as certain reflex levels are known, the diagnosis of nervous diseases becomes simple.

For example, if one draws a cross-section of the spinal cord, it is seen to consist of two divisions, the white matter and the central gray, which in turn possess an anterior and a posterior horn. The motor fibers originating in the anterior horn cells emerge from the anterior horn and the fibers subserving sensation originating in the posterior root ganglia enter the posterior horn, to ascend in the posterior column of the same side as do sensations for joint and muscle sense and touch and in the opposite spinothalamic tract. The motor and sensory roots with their connections in the gray matter constitute the lower reflex arc. It is necessary to remember only the location and function of three particular columns in the spinal cord: the lateral, consisting of the descending pyramidal tract fibers; the posterior, carrying deep sensation, and the spinothalamic, carrying sensations of pain and temperature.

If a peripheral nerve is severed, one, of course, expects to find loss of motor, sensory and trophic supply, and as the reflex arc has been severed, an absence of deep reflexes at that particular level. When that occurs, one may be dealing with an injury of a peripheral nerve, a multiple neuritis or compression of the nerve by a tumor. If only motor and trophic functions are lost, the lesion must be situated either in the anterior horns or in the anterior roots and may be only an acute or chronic anterior poliomyelitis, progressive muscular atrophy. If, in addition to the anterior horn, the lesion involves the commissures of the gray matter, the fibers for pain and temperature sense are interrupted as they cross, and there is in addition an analgesia, but a preservation of the sense of touch. This can be only a syringomyelia or a tumor of the medullary cord. As the pyramidal tract is part of the upper motor neuron, and in addition to subserving motion inhibits the lower reflex arc, a spastic paralysis with increased deep reflexes occurs when it is destroyed. If, therefore, a spastic paraplegia exists without loss of sensation, it can be only a lateral sclerosis which may be primary, as Erb's spinal spastic paralysis, or the beginning of a system disease involving other tracts later. If in addition to the spastic paraplegia there is an atrophic flaccid paralysis of the upper extremities without sensory loss, it can mean only a simultaneous involvement of the lateral columns and of the anterior horns, or an amyotrophic lateral sclerosis. If in addition to the spastic paraplegia there is loss of deep sensation with accompanying ataxia, it can be due only to a simultaneous involvement of the posterior and lateral columns, a posterolateral sclerosis or a combined degeneration of the cord usually due to pernicious anemia. If there is only a loss of deep sensation with resulting ataxia and evidence of a meningeal involvement about the posterior roots, shown in absent deep reflexes and pain, one is dealing only with tabes dorsalis or locomotor ataxia.

It therefore is evident that if only these few facts are remembered, the diagnosis of nervous diseases becomes so simple that even orthopedic surgeons make it.

EPILEPSY IN CHILDREN. DR. HUGH T. PATRICK.

This paper was published in full in the April issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN (**35**:653, 1928).

INTRACRANIAL TUMORS OF CHILDHOOD. DR. LOYAL DAVIS.

Attention was called to the syndrome which is commonly found in the tumors which arise from the roof of the fourth ventricle and which lie immediately above the medulla. These tumors, microscopically, are of the glioma group and are classified as medulloblastomas. The pathologic differentiation was made between these embryonic growths, which arise from the medulloblasts, and the more benign ependymomas, which arise from the ependymal cells lining the cavity of the fourth ventricle.

The most striking clinical symptoms which are presented by the medulloblastomas are headaches, which are usually suboccipital in location; persistent vomiting; early signs of a secondary internal hydrocephalus, tilting of the head and papilledema.

The surgical difficulties encountered in the treatment of patients with these tumors was emphasized as being due to their precarious situation and their embryonic nature.

DISCUSSION

DR. ISAAC A. ABT: I think I express the feeling of every one present in saying that we owe a great debt of gratitude to the officers of the Society who arranged this excellent symposium. I think we are unfortunate in that our respected colleague, Dr. Patrick, did not join us in the pediatric field, for if he had done as much for pediatrics as he has for neurology, he would have been a great addition to our ranks. We are fortunate to have heard Dr. Patrick, for he has been a teacher of neurology to students and practitioners for many years.

As I listened to these papers, my principal reaction was that the pediatrician is a general practitioner, a borderline man, who should know not only all about green stools and curds, and perhaps infant welfare, but a great deal about general diagnosis. This point should be much emphasized to the younger men. If I were asked to tell a young man how best to prepare himself for pediatrics, I would say that he should spend a great deal of time in a clinic for diseases of the heart and in a neurologic clinic, and should then come back to the pediatric fold.

Referring to Dr. Patrick's paper, I think we as pediatricians are greatly interested in convulsions in the infant. The question we always have to answer is: What is the significance of the attack? Are these little spasms spasmophilic, or are they due to some organic disease of the brain, the effect of an infantile cerebral palsy; have they the characteristics of a future epilepsy; are they due to some simple toxic effect of an acute infectious disease or possibly to a chronic disease? It seems to me these are the problems in differential diagnosis that we must always study. The question is: Is this baby with convulsions a potentially epileptic person? This is sometimes difficult to decide.

I was much impressed with what Dr. Patrick said about treatment. As he pointed out, many believe that these children require little if any treatment and that they must have their attacks when they come, but I fully agree that they must be treated persistently with either bromide or phenobarbital after the diagnosis has been made. Repeated attacks of convulsions cause

the expenditure of nerve energy and produce mental and physical deterioration. The lesser evil is to cause a cessation or reduction in the number of convulsions.

DR. JOSEPH BRENNEMANN: I agree with Dr. Abt in all his remarks, as one usually can. From an immediately profitable clinical standpoint, Dr. Patrick's paper was most interesting. I rather agree with him that most physicians should be "shot, hanged, thrown into the river, jailed or quartered," as he said, because they do not treat these patients as long and intensely as they should. I have always had the idea that petit mal is a serious thing, but it is usually not taken so seriously, and I have wondered whether it was advisable or productive of results to do much early. Dr. Patrick has not told us what results he gets when he treats these children as they should be treated.

I would like to have Dr. Patrick tell us what he means by "giving enough bromide" and "not being afraid of it." How much, how often and how continuously? If a child does not have an attack for a year, would he give bromide all the next year? Does he ever get unfavorable results, especially the severe skin lesions the dermatologists tell us about, that may last for years afterward, as I saw in one case?

DR. ALBERT B. YUDELSON: An important factor in these cases is the careful taking of the birth history. The physicians should know whether the child is the first born, the character of the labor, whether or not it was protracted and whether or not forceps were applied.

As far as dizzy spells are concerned, in early adult life, when these patients have headaches that are periodic, whether or not they are accompanied by vomiting, the question of migraine should be considered. Not infrequently attacks of petit mal occur in migrainous cases.

It is a question whether convulsions in early childhood do not constitute an open door to epilepsy in the future.

It must be remembered that dizziness and syncope on exertion not infrequently occur in chronic endocarditis, and differentiating this from attacks of petit mal should be the aim of the examination.

DR. A. H. PARMELEE: I would like to have Dr. Pollock tell us what he considers the significance of the Chvostek sign. Has it any particular pathognomonic significance? We see it in spasmophilia, in various types of meningitis, and following convulsions of various types. I wonder whether it simply means a lowered threshold to stimulus, or whether it has some other significance.

Another thing I think deserves consideration is what Dr. Davis said in regard to the possibility of sudden death from spinal punctures, not only in tumors of the brain but in cases of hydrocephalus or in other disturbances of the mechanics of the central nervous system. I had this so strongly impressed on me during my internship that I shall never forget it. During this time in the Kansas City General Hospital there was an epidemic of meningitis in the southwest, and in 1913 I reported 250 cases of epidemic meningitis. Of that series, ten patients died suddenly immediately following spinal puncture. Any one who has seen a person die under these conditions will not soon forget it. The cause of these sudden respiratory failures was probably the changes in intracranial mechanics incident to the withdrawal of cerebrospinal fluid. It is well to call attention, from time to time, to the fact that the simple procedure of lumbar puncture is not entirely without danger.

DR. JESSE R. GERSTLEY: Is there any difference in the prognosis of the type of epilepsy in which the attacks occur exclusively at night and the ordinary type, and is there any explanation for the fact that attacks occur when the patient is asleep?

DR. C. A. ALDRICH: I wish to know whether Dr. Patrick has noticed any of these attacks in the form of cardiac palpitation, and how he would differentiate between attacks of petit mal and neurasthenic symptoms.

I would also like to have Dr. Davis tell us whether he has ever tried radium emanations for these gliomas.

DR. HENRY E. IRISH: Has the so-called ketogenic diet or starvation treatment we have seen mentioned so frequently in the pediatric journals achieved a place among neurologists as part of the treatment of epilepsy?

In the discussion of lesions of the brain Dr. Davis did not mention tuberculoma. Starr, in reporting a series of 300 collected cases, mentioned tuberculoma as the most frequent tumor of early childhood, I think he said that it comprises over 51 per cent of these tumors. I would like to have Dr. Davis tell us his experience.

DR. LEWIS J. POLLOCK: The Chvostek sign, I think, is only an expression of increased irritability of muscle that is demonstrated by other methods as well, such as direct myotatic irritability, myodema or the muscular reactions to the electrical currents in which the muscle contracts to minute dosages of the anodal closure stimulations. These conditions are found in many states and occur commonly in both spasmophilia and tetany, which are closely related physiologically, and are probably due to some form of alkalosis.

DR. HUGH T. PATRICK: The relation of infantile convulsions to epilepsy has not been finally settled. In a paper by Dr. Levy and myself, published in *The Journal of the American Medical Association*, that question is as nearly answered as it ever has been. I think it really does throw light on certain types of infantile convulsions and epilepsy.

How long and in what dosage to continue the use of bromide is also an unanswerable question, but I shall make a stab at it. I am accustomed to tell these patients that they must continue treatment constantly for three years after the last attack. The attacks of petit mal are fully as difficult to control as fullblown epilepsy. The dosage is to be gaged by two things: what the attacks require and what dosage the patient can stand. There are many cases of epilepsy in which the patients cannot be cured by bromide, but I am talking about diagnosing the condition and treating the patient early. I have not seen many unfavorable results from the use of bromide. I do not claim much erudition, but I do claim a fair amount of common sense, and there is no sense in stupifying a patient for weeks and weeks. On two or three occasions, I have produced an ulcer which never progressed far if the patient followed the proper treatment.

The question is: How much can the child stand? Children tolerate bromide well, but the physician must find out how much each individual patient can stand. Any one can answer this as well as I. Ordinarily a child can take bromide three times a day for three years without any bad effects. It is not the bromide that causes weak-mindedness and idiocy, etc.; it is the epilepsy or the child's brain that does it.

I cannot teach you the difference between petit mal and grand mal, but in the little attacks that come without ascertainable cause, that are transient and that are related to nothing else, if the child is understood properly as an individual, the diagnosis is not so difficult. These little things in the way of forerunners are not difficult to diagnose if one is on the qui vive and does not ignore them.

I think the prognosis in nocturnal epilepsy is not different from that in any other form. Why some patients have nocturnal attacks and no others for long periods I do not know, and I have never known any one who did. Generally, after a time such a patient has also diurnal attacks.

How can a physician differentiate petit mal from palpitation? How can he tell typhoid from pneumonia? The child does not have neurasthenia; I have never seen a case.

I know nothing about the ketogenic diet, and have never tried it. A man who has been treating patients with epilepsy for more years than he cares to expose has tried and has seen tried a long list of things, and he gets tired of trying new things. Whether it is because I have grown lazy or skeptical,

or whether I think the other fellow is barking up a vacant tree, I let him try the new things. If any one reviews the list of things that have been advocated by good men, by serious students, by research workers and by gastro-enterologists, and then finds the bulk of these things long lost and forgotten, he is a little slow about taking up new treatments. I would not condemn this treatment — I just do not know.

Dr. Loyal Davis: I have never used radium emanations in the treatment of gliomas. Our few experiences in using radium were disappointing and unfortunate. In all probability, the fault was ours in that we were inexperienced in controlling the dosage.

I mentioned the occurrence of tuberculomas in childhood, but their frequency is small as compared with medulloblastomas. When the cases of Dr. Allen Starr were reported, the microscopic study of tumors of the brain was not all that it should have been, and tuberculosis in general was more prevalent. Further, a tuberculous meningitis is such a common occurrence in tuberculomas of the central nervous system that it may account for the rarity with which they present themselves in a neurosurgical clinic as tumors per se.

From a surgical standpoint, when a tuberculoma is found on operation, the prognosis is much better if it is left untouched and if general hygienic measures, together with Rollier's treatment, are instituted.

ST. LOUIS PEDIATRIC SOCIETY

Feb. 3, 1928

T. Wistor White, M.D., *President*

Dr. E. W. Saunders: A Sketch of His Work and Character. Dr. John Zahorsky.

Dr. E. W. Saunders was an aggressive Christian, an unyielding fundamentalist, and yet an enthusiastic man of science. He regarded medical theories as temporary working hypotheses, and never permitted scientific researches to change his Christian faith.

For fifty years he practiced medicine in St. Louis; his work was finally restricted to pediatrics. He was a splendid teacher and a kind and generous friend to the young physician. Medical research continued to engage his attention in all his practice, but most of his work has been lost to literature. He was a member of the American Pediatric Society and had many friends throughout the United States. He was a hard-working student, an old time classical scholar and a gentleman of the most excellent southern type.

Essential Pentosuria. Dr. J. A. Rossen.

The case presented was that of a boy, aged 13, who had been treated for a year and a half as a diabetic patient. In spite of diet, his urine continued to show small amounts of a reduction substance. The patient gave a history of having gained in weight but presented no other symptoms of diabetes mellitus, with the exception of the reduction substance in the urine. Results of physical examination were negative. He was sent to the Jewish Hospital with a tentative diagnosis of renal glycosuria. His blood sugar and sugar tolerance were normal. His Wassermann reaction was negative. The reduction substance in the urine did not ferment with yeast and gave a positive reaction to the Bial test; the melting point also corresponded with that of pentozazone. The final diagnosis was essential pentosuria. The case was presented to call attention to the possibility of mistaken diagnosis and the necessity for making complete tests of reduction substances found in the urine.

HYPERTROPHIED THYMUS IN THE NEW-BORN. DR. J. A. ROSSEN.

A boy, weighing 3,000 Gm. was born Nov. 2, 1927, with marked asphyxia, the cyanosis being extreme. It required thirty-five minutes to resuscitate the child. When first seen by a pediatrician at 3 p. m., there was still marked cyanosis of the extremities and face. The anterior fontanel was not tense. A loud systolic murmur was heard all over the heart. The impression at that time was that the child had a congenital defect of the heart. A roentgenogram was ordered. Ten cubic centimeters of the mother's blood was injected into the muscles. The infant continued to have cyanosis and periods of apnea. Alphalobeline was given. The roentgenogram taken when the infant was 6 hours of age showed a shadow that almost entirely filled the chest cavity. On the third day the anterior fontanel seemed tense. Roentgen-ray treatment was given to the thymus area. The infant showed remarkable improvement. Another picture taken seven days later showed a shadow of a normal thymus. The infant has continued to do well and has not had further symptoms. The final diagnosis was hypertrophied thymus.

TWO CASES OF CHLOROMA. DR. J. A. ROSSEN, and DR. J. KNOTT.

The authors presented gross and microscopic specimens from a case of chloroma in a girl, aged 3 years, whom they had observed during September, 1926. They also presented a living patient, a girl, aged 10, who had a large tumor of the right eye with a typical myelogenous blood picture. A section of the tumor showed that it was made up of the same type of cells as those found in the blood. The patient was treated with radium at the Barnard Skin and Cancer Hospital, and there was a remarkable reduction in the size of the tumor of the orbit. A secondary anemia increased and transfusions were resorted to. In spite of the fact that the patient has been receiving radium and the tumor of the orbit has become smaller, a new tumor over the occiput made its appearance during the last few days before presentation of the patient. It seems to be growing rapidly. Drs. Rossen and Knott did not have any suggestions to offer concerning treatment, except to recommend the use of radium. Irradiation of other parts of the body and its effect on the blood picture is being checked.

NEW YORK ACADEMY OF MEDICINE, SECTION OF PEDIATRICS

Feb. 9, 1928

WILLIAM ROPES MAY, M.D., *Chairman*

THE KETOGENIC DIET IN EPILEPSY. DR. FRITZ B. TALBOT, Boston.

The action of the ketogenic diet in epileptic children has proved sufficiently beneficial to warrant the use of this type of therapy. Various investigators have demonstrated that the effectiveness of the diet depends neither on lowering of the blood sugar content nor on shifting of the acid-base equilibrium per se unless some other factor is involved. The hypothesis that the anesthetic action of acetone is responsible for the beneficial effect seems more probable. Long continued use of this apparently abnormal diet by epileptic patients is usually associated with constitutional improvement and increased resistance to all types of disease. Because small aberrations from the ketogenic regimen retard improvement disproportionately, it is essential that the diet be prescribed in great detail and administered by a person with special training.

The shifting from a normal mixed diet to a ketogenic diet of the desired ratio can be made gradually by increasing the intake of ketogenic substances and simultaneously decreasing the intake of antiketogenic foods. An intermediate

period of fasting is not necessary in most cases. As the response of different patients to the diet varies greatly, certain tests should be made to ascertain the degree of ketosis evoked.

In planning the ketogenic diet, it is essential to remember that the average child needs about 50 per cent more than the calculated basal caloric requirement for a person of the same height and age, but what personal variations in activity make it impossible to determine the correct intake without careful checking. There has been a great deal of debate about the minimal protein requirement, but nothing definite is yet known about the exact quantity of exogenous protein used by the body. At the Massachusetts General Hospital it has been found that when the total intake of food is adequate, 1 Gm. of protein per kilogram of body weight appears to be sufficient for children of all ages before ketosis becomes marked. During ketosis the nitrogen balance of children more than 8 or 10 years of age is not positive until the protein intake is about 1.25 Gm. per kilogram of expected body weight. This suggests that the amount of protein in the diet should be larger than has previously been thought necessary. No available evidence indicates the damage done to the body by persistence of a negative nitrogen balance, but it is reasonable to suppose that an indefinite quantity of protein cannot be lost without injury of some sort. Consequently, it is desirable to determine the balance from time to time during dietary therapy and almost essential to do so when the diet is extreme in any way. The nitrogen excretion, however, varies so much from day to day, even when the intake remains constant, that significance can be given only to figures showing the average excretion for a period of three days or more.

Certain factors other than the action of carbohydrate and fat seem to affect the retention and availability of nitrogen in the body. What they are is not known.

Until the mineral metabolism of the body is more thoroughly understood, it seems desirable to give small doses of calcium lactate (2 to 3 Gm. daily) during prolonged ketosis, both because it is impossible to include adequate quantities of calcium in a diet high in fats and because ketosis increases the excretion of calcium.

THE KETOGENIC DIET. DR. M. G. PETERMAN, Milwaukee.

The ketogenic diet was introduced in 1924 after two years of clinical trial. In 1921, Wilder, on the basis of Geyelin's results with starvation, suggested that diets, in which the proportion of carbohydrate and protein were sufficiently restricted, might offer a method of treatment of epilepsy. It was on the basis of Wilder's suggestion that the high fat, low carbohydrate, restricted protein diet was evolved. Studies of patients on this diet and modifications of my original procedure have been reported by me during the past three years. Other investigators have reported results in the treatment of patients who had epilepsy with the ketogenic diet, but there have not been any modifications of my procedure, nor have there appeared any other original explanations of the action of the diet in its control of epileptic seizures.

In the preliminary report, it was suggested that the object of the diet was to produce acidosis. This object was based on the results reported in 1921 by Jarloev and by Geyelin. Geyelin was the first in this country to report the beneficial effects of fasting on epileptic patients, and he ascribed the results to the acidosis produced. Three years later, Hoeffel and Moriarty, and Shaw and Moriarty subscribed to the acidosis theory of Jarloev and Geyelin. In 1925 and subsequently, it was demonstrated that acidosis alone or ketosis alone do not control the convulsions of epilepsy.

Children on the ketogenic diet may be given enough soda to alkalize the urine without provoking attacks. The ketosis is variable during this procedure, but usually persists while the acid base equilibrium is shifted toward the alkaline. A chemical acidosis produced by the administration of ammonium chloride in large amounts does not control the convulsions of epilepsy. With this drug,

the carbon dioxide combining power of the blood may be reduced to 20 volumes per cent and the hydrogen ion concentration of the urine to 5.4. The fat in the diet may be substituted with intarvin until the acetone bodies disappear from the urine or are considerably diminished and still the patient be kept free from attacks. The effect of pyruvic acid, the incomplete oxidation product of intarvin, is not known. Authors should be more careful to distinguish between acidosis and ketosis. It has been demonstrated that the ketogenic diet produces a ketosis in which the acetone bodies may be increased from two to forty times normal. This increase is evident in the blood, urine and alveolar air and probably throughout the tissues. There is usually a slight fall in the alkali reserve or a decrease in the carbon dioxide combining power of the plasma, but not below the limits of normal. Talbot and his co-workers in their report still favored my original acidosis theory, but in their conclusions they agree with my subsequent ideas on the predominant effect of the ketones. McQuarrie and Keigh have recently subscribed to this proposal. They have also further substantiated my earlier demonstration that the degree of ketosis is a factor in the control of epileptic seizures. The fall in blood sugar is not always great or consistent. The normal variations of fasting blood sugars is well known. In ketosis the level is dependent on a number of factors, including the storage of glycogen before ketosis, the degree of ketosis, the total caloric intake, individual susceptibilities and the rate of absorption. Brown and Graham, McQuarrie and Keith and others do not find a consistent decrease. There is no change in the calcium, phosphorus, chlorides or nonprotein nitrogen of the blood, nor in the number of blood cells or sedimentation reaction. The effects of the ketogenic diet as outlined in the foregoing paragraph are not, as suggested by McQuarrie and Keith, "entirely similar to those of fasting." It has been sufficiently demonstrated that in ketosis the acetone bodies are increased in a much higher proportion and that they persist for longer periods. There is also not the marked decrease in the carbon dioxide combining power of the plasma nor the change in the hydrogen ion concentration of the blood.

NORTHWESTERN PEDIATRIC SOCIETY

Winter Meeting, Feb. 17, 1928

RAY SHANNON, M.D., *President, in the Chair*

MECKEL'S DIVERTICULUM. DR. RAY SHANNON.

Meckel's diverticulum, while perhaps rare, has not attracted the attention as a cause of disease in infancy and childhood that it deserves. The symptoms that it produces are often obscure and difficult of diagnosis and at the same time are frequently serious in their effect on the patient. They may be roughly divided into three classes: first, mechanical; second, inflammatory, and third, ulcerative. With the mechanical symptoms may be included, besides obstruction, intussusception due to an inversion of the Meckel's diverticulum. The inflammatory symptoms may arise from an inflammation of the diverticulum similar to that of an appendicitis. Ulcerative symptoms may be those of hemorrhage or of perforation, either acute or chronic.

Two cases were presented; the first was that of an infant girl, aged 4 months, who presented early the symptoms of a simple gastro-intestinal disturbance as a result of an infection of the upper respiratory tract. The small amount of blood found in the stool was unexplained but was thought to be due to a gastro-enteritis. After a mild course of three or four days, the infant suddenly became violently ill with high temperature, extreme distention, vomiting and prostration. Operation revealed an intussusception wholly within the ileum, with gangrene of the intussuscepted bowel. Death occurred shortly after the operation. The intussusception was shown to be due to an inverted

Meckel's diverticulum. The second case was that of a girl, aged 16 months, who was seen because of extensive hemorrhage from the bowel and who did not have pain or a high temperature. Repeated transfusions were necessary to prevent exsanguination. The early diagnosis was probably a bleeding duodenal ulcer, but when the hemorrhage recurred twice during a period of two weeks, while the patient was under treatment, a diagnosis of Meckel's diverticulum was made and operation was performed. A Meckel's diverticulum was found with ulceration at the base and chronic perforation with localized peritonitis. Removal of the diverticulum was followed by complete and rapid recovery.

The symptoms of Meckel's diverticulum are often obscure and atypical. It is therefore felt that an intimate knowledge of this condition in infancy and childhood will permit greater boldness in advising surgical intervention in the abdominal conditions of this age.

THREE CASES OF CARDIO SPASM OF THE NEW-BORN WITH ROENTGEN-RAY STUDIES. DR. BIRNBERG.

This article will be published in full in a later issue.

CONGENITAL HEMOLYTIC ICTERUS. DR. HEDENSTROM.

A girl, born at term on May 22, 1925, had copper colored skin which gradually faded toward the end of a year. The father was 35 years of age, and the mother was 38; both were living and well. There were three other children living and well. The paternal grandmother had skin of a slight icteric hue.

The first examination, at the age of 16 months and 22 days, revealed an undernourished child, weighing 19½ pounds (8.8 Kg.). The temperature was 99 F. The patient was unable to stand or walk. Marked pallor, icterus of the skin and bulbar conjunctiva were present. The fontanel was enlarged. Ten teeth were erupted. The spleen was readily palpable 4 cm. below the costal margin. The stools were brown but were never clay colored. The blood Wassermann reaction and the Pirquet test were negative; the urine was normal. The fragility was normal; hemolysis began at 0.45 and ended at 0.35. The blood picture showed an anemia of 2,100,000 erythrocytes; hemoglobin, 40 per cent; leukocytes, 5,000; polymorphonuclears, 33 per cent; eosinophils, 12 per cent and basophils, 3 per cent. There was marked anisocytosis, polychromatophilia, slight poikilocytosis, a moderate number of normoblasts and a definite diminution in the size of the red corpuscles.

Development of an infection of the upper respiratory tract resulted in a poor condition with hemoglobin 15 per cent; erythrocytes, 840,000; leukocytes, 30,000 and 4 normoblasts per hundred leukocytes. There was marked anisocytosis, poikilocytosis and polychromatophilia.

Following two citrated blood transfusions, the blood picture improved; the hemoglobin increased to 54 per cent, and the erythrocytes numbered 21,920,000.

Splenectomy was performed; the spleen weighed 134 Gm. Without further transfusion, steady improvement followed. Ten months after the operation, the hemoglobin had increased to 90 per cent and the erythrocytes to 4,200,000, and the blood morphology was normal. The child has learned to walk and to run about and is now apparently normal.

MILROY'S DISEASE. DR. A. V. STOSSER.

This article will be published in full in a later issue.

THE HYPERTROPHIES OF INFANCY AND CHILDHOOD: CONGENITAL INTERPULMONIC CYST. DR. SWANSON and DR. E. S. PLATOU.

This article will be published in full in a later issue.

Book Reviews

KLINISCHE UND SEROLOGISCHE BEITRAGE ZUM SCHARLACHPROBLEM. G. FANCONI. Pp. 66. Berlin: S. Karger, 1926.

The author of this monograph reviews various phases of scarlet fever and discusses the changes in the blood—especially in the leukocytes—the variations in immunity during convalescence, superinfection in convalescence, recurring eruptions, scarlet fever and injections of serum, scarlet fever and chickenpox, scarlet fever and vaccination against smallpox, scarlet fever and measles and other febrile diseases. He devotes considerable space to serologic differentiation of scarlet fever, the blanching phenomenon, streptococci in the diagnosis of scarlet fever, scarlet fever and scarlatinoid conditions.

Fanconi favors the view that scarlet fever may be caused by a variety of different agents. He holds that even at the present time the principle of a specific cause of scarlet fever has not been definitely established, and that even if a particular agent is the cause of most cases of scarlet fever, there are other agents that can call forth the symptoms of scarlet fever with striking completeness. Consequently, the problem of scarlet fever cannot be solved fully by searching exclusively for a single specific agent. The body of the patient and its reaction to possible causes should not be overlooked. The one-sided overemphasis on bacteria, which so thoroughly influenced the study of medicine during the period of the great bacteriologic discoveries, now belongs to medical history.

DES KINDES, ERNÄHRUNG, ERNÄHRUNGS-STÖRUNGEN UND ERNÄHRUNGS THERAPIE. By A. CZERNY and A. KELLER. Second edition. Volume 2. Part 2. Price 36 R. M. Leipzig: Franz Deuticke, 1928.

This is the last of the second edition of this monumental work; the first edition began to appear in 1905, and was completed about 1912.

Such an exhaustive book on this subject has never before been written. Not only is the literature covered so completely that it is safe to say that no important articles in any language have been missed, but all points of view and opinions are reviewed and the clear light of expert criticism turned on them. The authors are not only critics, but the result of experience, study and thought is given in their opinions and advice in a lucid direct way that calls forth admiration whether one agrees with their views or not.

It is especially pleasing to Americans that the book displays such knowledge and recognition of our scientific efforts on nearly all subjects discussed; this is not often met with in the foreign literature.

The portion of the work under review deals exhaustively with nutritional disturbances in constitutional disorders such as exudative diathesis, tetany, rickets, epilepsy, anemia and other conditions. Then a section on nutritional disturbances in congenital deformities; megacolon, congenital atresia of the bile ducts, myxedema and diabetes mellitus.

Following this section is a most complete discussion on metabolism covering nearly 450 pages.

The book may be read with pleasure and profit, but as a reference book it probably has no equal.

BEITRAGE ZUR KENNTNIS DER MONGOLOIDEN MISSBILDUNG (MONGOLISMUS AND MONGOLOIDE IDIOTIE). By DR. W. M. VAN DER SCHEER. Pp. 156. Berlin: S. Karger, 1927.

This book deals almost entirely with 348 authentic cases of mongolian idiocy. Underlying the treatise is an apology to the mongolian race. The main contention is that mongolism is not a retroversion, but that it is a

malformation in which the median skull defects overshadow the picture. The congenital defects can all be traced back to a narrow amniotic sac which produces the greatest damage from the sixth to the seventh week of embryonic development. The disturbances in the amnion occur due to a poor implantation of the ovum on a normal endometrium. The metabolic, the trophic and psychic disturbances as they occur after birth are due to a hypoplasia of the brain and poor development of the autonomic nervous system.

Much consideration is given to the views of other authors on the subject, and the treatise is most complete and is presented in an interesting manner. The portions on the differential diagnosis from other mental defects and cretinism and on the etiology are especially attractive.

The book should be of value not only to the pediatrician but to the obstetrician, the oculist, the laryngologist, the dentist, the anatomist and the psychiatrist.

LE SYNDROME MALIN. By V. HUTINEL, M.D. Pp. 305. Price, 32 francs. Paris: Masson et Cie, 1927.

This book is a development of the clinical lectures given by the author on a rather unique phase of clinical pathology. He has observed in his years of practice a certain set of clinical symptoms, namely prostration, nervous reactions, circulatory failure, erythema, high temperature and signs of passive congestion. These are found in eruptive fevers, typhoid, diphtheria, pneumonia, enteritis and other severe general diseases. As this syndrome is always found in the most severe of these conditions, the author believes that it should be classed as a distinct syndrome of a common origin. He thinks that this origin may be anaphylactic.

The book is divided into six chapters. The first four contain a complete discussion of this syndrome in the diseases mentioned. In the fifth chapter the origin of the syndrome is discussed, and in the last chapter is given the author's results in hospital treatment. The volume is well written and organized.

PRECIS DE MEDECINE INFANTILE. E. WEILL and G. MOURIQUAND. Fourth edition. Price, 90 francs. Pp. 1195, with 178 illustrations. Paris: Gaston Doin et Cie, 1928.

This is a general textbook of pediatrics for the practitioner. The French ideas as to diagnosis and therapy are well presented, and the section on respiratory diseases is especially good. The illustrations are well chosen to show the conditions discussed, but are not of such excellence as one is accustomed to see in German and American texts.

If one wishes a complete textbook in one volume giving the French point of view and practical approach, this book is well worth while.

Directory of Pediatric Societies

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION OF DISEASES OF CHILDREN

Chairman: Dr. Hugh McCulloch, 500 S. Kingshighway, St. Louis.
Secretary: Dr. C. A. Aldrich, 545 Lincoln Ave., Winnetka, Ill.
Place: Minneapolis. Time: June 11-15, 1928.

AMERICAN PEDIATRIC SOCIETY

President: Dr. J. Claxton Gittings, 1828 Pine St., Philadelphia.
Secretary: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Place: Washington, D. C. Time: April 30 to May 2, 1928.

THE ASSOCIATION OF AMERICAN TEACHERS OF THE DISEASES OF CHILDREN

President: Dr. J. P. Crozier Griffith, 1810 Spruce St., Philadelphia.
Secretary: Dr. Paul W. Emerson, 86 Bay State Road, Boston.
Place: Minneapolis. Time: June 12, 1928.

AMERICAN CHILD HEALTH ASSOCIATION

President: Hon. Herbert Hoover, 370 Seventh Ave., New York.
Secretary: Dr. Philip Van Ingen, 370 Seventh Ave., New York.
Place: Stevens Hotel, Chicago. Time: October 15-19, 1928.

CANADIAN SOCIETY FOR THE STUDY OF DISEASES OF CHILDREN

President: Dr. G. R. Pirie, 20 Madison Ave., Toronto.
Secretary: Dr. A. P. Hart, 1844 Bloor St. West, Toronto.
Place: Vancouver, B. C. Time: June 29-30, 1928.

SECTIONAL

CENTRAL STATES PEDIATRIC SOCIETY

President: Dr. H. T. Price, 121 University Place, Pittsburgh.
Secretary: Dr. A. H. Parmelee, 310 S. Michigan Ave., Chicago.

SECTION ON PEDIATRICS OF SOUTHERN MEDICAL ASSOCIATION

Chairman: Dr. E. C. Mitchell, 1073 Madison Ave., Memphis, Tenn.
Secretary: Dr. A. J. Waring, De Renne Apts., Savannah, Ga.
Place: Asheville, N. C. Time: November 12-15, 1928.

LOCAL

NORTHERN CALIFORNIA PEDIATRIC SOCIETY

President: Dr. W. L. Channell, Medical Bldg., Oakland, Calif.
Secretary: J. E. Parrott, Medico-Dental Bldg., San Francisco.
Place: Various Teaching Schools. Time: Monthly.

SOUTHWESTERN PEDIATRIC SOCIETY

President: Dr. Oscar Reiss, Westlake Professional Bldg., Los Angeles.
Secretary: Dr. William Sidney Bowers, 1136 W. 6th St., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

THE ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. W. Wiley Jones, Majestic Bldg., Denver.
Secretary: Dr. Berryman Green, 537 Republic Bldg., Denver.
Place: Children's Hospital, Denver. Time: Third Saturday of each month, 2:30 p. m., September to May, inclusive.

GEORGIA PEDIATRIC SOCIETY

President: Dr. Joseph Yampolsky, 127 Peachtree St., Atlanta.
Secretary: Nevin M. Atkins, Atlanta.
Place: Savannah, Ga. Time: May 9, 1928.

PEDIATRIC SECTION OF FULTON COUNTY MEDICAL SOCIETY, ATLANTA, GA.

President: Dr. William W. Anderson, 362 Peachtree St., Atlanta.
Secretary: Dr. Lee Bivings, Exchange Bldg., Atlanta.
Place: Academy of Medicine, 32 Howard St., Atlanta.
Time: Second Thursday of each month from October to April, at 8 p. m.

NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. James Herbert Young, 66 Commonwealth Ave., Boston.
Secretary: Dr. Thomas Lanman, 286 Marlborough St., Boston.
Place: Boston Medical Library. Time: Second Friday of each month.

CHICAGO PEDIATRIC SOCIETY

President: Dr. Maurice Blatt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. J. K. Calvin, 104 S. Michigan Ave., Chicago.
Place: City Club. Time: Third Tuesday of each month.

LOUISIANA STATE PEDIATRIC SOCIETY

President: Dr. L. R. De Buys, 1487 Delachaise St., New Orleans.
Secretary: Dr. F. J. Kinberger, 3601 Prytania St., New Orleans.

DETROIT PEDIATRIC SOCIETY

President: Dr. George E. White, Capitol Theatre Bldg., Windsor, Ontario, Canada.
Secretary: Dr. W. S. O'Donnell, 1563 David Whitney Bldg., Detroit.
Place: Children's Hospital of Michigan. Time: No fixed date; approximately once a month.

NORTHWESTERN PEDIATRIC SOCIETY

President: Dr. F. W. Schlutz, University of Minnesota, Minneapolis.
Secretary: Dr. E. S. Platou, 730 La Salle Bldg., Minneapolis.
Place: University Campus and Rochester. Time: Winter, Spring, Summer, Fall.

ST. LOUIS PEDIATRIC SOCIETY

President: Dr. Park J. White, Beaumont Bldg., St. Louis.
Secretary and Treasurer: Dr. Ralph L. Cook, 508 N. Grand Blvd., St. Louis.
Place: St. Louis Children's Hospital. Time: At least once a year and contingent thereafter on the arrangement of a suitable program.

NEBRASKA-IOWA PEDIATRIC SOCIETY

President: Dr. Floyd Clarke, 731 City Nat'l Bank Bldg., Omaha.
Secretary: Dr. R. B. Eldredge, 1036 Medical Arts Bldg., Omaha.
Place: Add-Sell Restaurant. Time: Second Thursday of each month from October to May, inclusive, 6 p. m.

BROOKLYN PEDIATRIC SOCIETY

President: Dr. Louis M. Ruderman, 961 Eastern Parkway, Brooklyn.
Secretary: Dr. Joseph F. Paulonis, 1357 Bushwick Ave., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

BRONX PEDIATRIC SOCIETY

President: Dr. William Hinz, 1964 Grand Concourse, New York.
Secretary: L. H. Barenberg, 1749 Grand Concourse, New York.
Place: Concourse Plaza, 161st, St., and Grand Concourse, New York.
Time: Second Wednesday of each month, except June, July, August and September.

CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady, N. Y.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany, N. Y.
Place: Various Cities in New York. Time: Fourth Thursday of October, January and April.

PEDIATRIC SECTION OF THE NEW YORK ACADEMY OF MEDICINE

President: Dr. Stafford McLean, 17 East 71st St., New York.
Secretary: Dr. Frank Elmer Johnson, 300 West End Ave., New York.
Place: New York Academy of Medicine, 2 East 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

THE PEDIATRIC SECTION OF THE ACADEMY OF MEDICINE OF CLEVELAND

Chairman: Dr. J. W. Epstein, 3912 Prospect Ave., Cleveland.
Secretary: Dr. Charles W. Burhans, 14805 Detroit Ave., Lakewood, O.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

PEDIATRIC SECTION, ACADEMY OF MEDICINE, TORONTO

President: Dr. M. A. Cox, 34 St. Clair Ave. West, Toronto.
Secretary: Dr. F. R. Hassard, 1361 King St. West, Toronto.
Time: November 17, December 15, February 16 and March 15.

PHILADELPHIA PEDIATRIC SOCIETY

President: Dr. Edward L. Bauer, Jefferson Medical College, Philadelphia.
Secretary: Dr. John Porter Scott, 2040 Pine St., Philadelphia.
Place: College of Physicians, 19 S. 22nd St., Philadelphia. Time: Second Tuesday of each month from October to June, inclusive.

MEMPHIS PEDIATRIC SOCIETY

President: Dr. W. D. Blue, 1024 Madison Ave., Memphis, Tenn.
Secretary: Dr. Gilbert J. Levy, 899 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

TEXAS PEDIATRIC SOCIETY

President: Dr. Edwin G. Schwarz, Medical Arts Bldg., Fort Worth.
Secretary and Treasurer: Dr. Ramsey Moore, Medical Arts Bldg., Dallas.
Place: San Antonio. Time: October, 1928.

RICHMOND PEDIATRIC SOCIETY

President: Dr. T. D. Jones, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. W. Ambrose McGee, West End Medical Bldg., 2618 Grove Ave., Richmond, Va.
Place: Westmoreland Club, 601 E. Grace St., Richmond, Va.
Time: First Thursday of each month (other than during the summer).

VIRGINIA PEDIATRIC SOCIETY

President: Dr. Lawrence T. Royster, University, Va.
Secretary: Dr. William B. McIlwaine, Petersburg, Pa.
Place: Danville, Va. Time: October 17, 1928.

NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. R. S. Miles, Fidelity Bldg., Tacoma, Wash.
Secretary: Dr. D. M. Dayton, 704 St. Helens Ave., Tacoma, Wash.
Place: Vancouver, B. C. Time: June 28, 1928.

SEATTLE PEDIATRIC SOCIETY

President: Dr. George B. McCulloch, Cobb Bldg., Seattle.
Secretary: Dr. C. L. Smith, Stimson Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, at 7 p. m.

MILWAUKEE PEDIATRIC SOCIETY

President: Dr. A. B. Schwartz, 418 E. North Ave., Milwaukee.
Secretary: Dr. M. G. Peterman, 167 Seventeenth St., Milwaukee.
Place: Children's Hospital, Milwaukee. Time: Second Wednesday of each alternate month, beginning with February.

INDEX TO VOLUME 35

The (*) preceding the page number indicates an original article. No author entries are made except for original articles. Book Reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B, O and S, respectively.

PAGE

Abdomen, acute, in children 348
surgical conditions in children 531
Abnormalities, amnion, free transplantation of upper extremity through amniogenic malformation 503
bilocular urinary bladder 504
chondrodystrophia fetalis 504
congenital attitude anomalies caused by muscular contractions: clubhand, wryneck and knee contractures 503
congenital defect of diaphragm 504
congenital encapsulation of small intestine. 503
dicephaly 502
Abscess, brain, in infants under 12 months of age; report of 2 cases with 17 collected cases *256
lung, bronchoscopic treatment of 527
lung, from the medical standpoint 316
lung, roentgen-ray diagnosis of 317
lung, surgical aspects of 333
multiple abscesses in newly born 304
peritonsillar, early tonsillectomy in 153
pulmonary, following tonsillectomy 329
Abt, I. A.: Survey of the communicable disease situation in Chicago *1048
Achondroplasia, fetal 300
Acid base equilibrium in blood of children .. 1112
in certain dermatosis 1137
in children with reference to intestinal disturbance 500
of plasma of infants with mastoiditis, effects of diarrhea, vomiting, dehydration and oliguria on *557
Acidosis, clinical value of phenomenon of, in digestive disturbance of infants 299
diabetic, and leukocytosis 517
Aciduria, orthostatic 1111
Acrodynia, infantile 333
juvenile 734
paroxysmal, of infancy 333
pathologic anatomy of 344
the acrodynic syndrome 324
Addison's disease, cured case 522
Aikman, J.: Shoe poisoning; report of 3 cases *1038
Albers-Schonberg's disease: See Osteosclerosis fragilis ..... .
Albumin, influence of, on proportion of some nitrogen containing substances in urine 670
Aldrich, C. A.: A new test for hearing in new born; the conditioned reflex ... *36
Alimentary tract, epithelial heterotopias of .. 134
Allergy, allergic manifestations in tuberculosis 732
Amaurosis: See Blindness ..... .
Amebiasis, therapy of 676

PAGE

Amnion, abnormalities, free transplantation of upper extremity through amniogenic malformation 503
Amyotonia congenita, pathology of 325
congenital; report of 2 cases 698
Anaphylaxis, anaphylactic factor in scarlet fever *991
heredity, transmission of protein hypersensitiveness from mother to offspring; passive sensitization in utero 904
Anemia, Biermer's, in childhood 695
congenital 732
extramedullary hematopoiesis in anemias .. 518
splenic, in infants 321
Aneurysm, treatment in a 6 year old boy .. 519
Angioma, of arm 157
ulcerated, of face 156
Anomalies, of new born 740
Anonychia congenita with loss of phalanges. 674
Anorexia, an analysis of *26
Antibodies, formation of, in nursling 668
Anus, atresia ani congenita, surgical treatment of 715
atresia ani perienalis 548
Apomorphine, paroxysmal tachycardia treated with 1130
Appendix, appendectomies in children 711
appendicitis lymphoid 541
appendicostomy, and dysentery 335
of child, in the roentgenogram 1139
Arrhythmia, auricular fibrillation in childhood; cases *420
auricular fibrillation in children 534
of childhood 1128
Arsenic, granulocytic aplasia of bone marrow following use of *1032
study of some organic arsenical compounds; their use in certain streptococcal infections 1139
Arsphenamine, comparative value of methods to reduce harmful effects of neoarsphenamine by different vehicles 313
interscalp method of administration of neoarsphenamine 723
neoarsphenamine in treatment of syphilis. 515
neoarsphenamine, treatment for congenital syphilis 514
sulpharsphenamine poisoning; case 313
Arteries, patent ductus arteriosus, hematoma of left suprarenal gland, meningeal hemorrhages and diverticulum *254
Arthritis deformans, polyarthritis deformans in children 1127
Ascaris, infestation, toxic meningitis and meningo-encephalitis due to 327
Asphyxia, resuscitation of apparently dead new-born infants by electric current ... 506
Asthma, administration of ephredine 143
clinic for study of eczema, hay fever and. 125
comparative methods of skin testing with differently prepared extracts of house dust 687
eosinophil cells in blood of normal and asthmatic children *171
in infants 321
nasal disease in relation to 706

PAGE

Atelectasis, acute massive, of lung.....*239
thymus enlargement as cause of..... 145
Athrepsia, and azotemia..... 900
definition of true athrepsia and its prognosis 299
prognosis in..... 338
Atrophy, pancreatic, in infancy..... 669
Atropine, action of, on milk secretion.... 298
influence of acidity on activity of homatropine and 340
Auricular fibrillation: See arrhythmia
Azotemia, and athrepsia..... 900

Baagøe, K. H.: Eosinophil cells in blood of normal and of asthmatic children...*171
Babies and children's hospital, inspection of. 734
Bacteria, bacillus bifidus.....1113
coccal forms of bacillus diphtheriae..... 497
Bakwin, H.: Sex factor in infantile tetany.*964
Bakwin, R. M.: Sex factor in infantile tetany *964
Barber, E. W.: Auricular fibrillation in childhood: report of cases.....*420
Barker, L.: Endocrine glands in relation to infancy and childhood.....*872
Bartlett, W. M.: An analysis of anorexia. *26
Bauer, E. L.: Celiac disease; cases.....*414
Behaviour, abnormal, physical problems of.*113
problems in children..... 541
study of, in new born..... 138
Bell, A. L. L.: Roentgenographic changes in thorax of normal new born babies; daily roentgenographic study of 25 normal babies during the first 14 days of life *837
Beriberi, production of, in nursing young, (mus norvegicus albinus) associated with hemorrhages.....*811
Bilirubin, normal content of serum..... 499
physiochemical properties of, in body fluids 668
solutions, decomposition of..... 499
Biographies:
Bagellardus, Paulus: ?-1492.....*289
Metlinger, Bartholomaeus.....*492
Oestereicher, Sebastian *894
Roelans, Cornelius; 1450-1525.....*659
Soranus of Ephesus *120
Victorius, Leonellus Faventinus, ?-1520; Knuffer, George, Jr.....*1106
Birth injuries, cause of death in new-born 138
cerebromeningeal lesions of new born dependent on labor..... 138
injury to liver at birth giving clinical picture of congenital absence of the biles ducts..... 301
intracranial, in new-born..... 505
of phrenic nerve causing paralysis of right diaphragm 138
Birth Rate, decrease in..... 494
Bismuth, treatment of congenital syphilis.. 681
Bladder, congenital diverticulum of urinary 136
tetany of sphincter of..... 325
urinary, bilocular..... 504
Blatt, M. L.: Pericarditis as a primary clinical manifestation of tuberculosis in childhood *631
Blindness, case of malarial amaurosis.....1135

PAGE

Blood, catalytic action of.....1112
effect of epinephrine on blood picture in normal, lymphatic and scrofulous children 159
easinophil cells in blood of normal and asthmatic children.....*171
groups, in children..... 668
group, hemagglutinins of mother and fetus 1116
hemoglobin resistance..... 297
occult, clinical significance of occurrence in feces 297
nature of derivates of blood pigment found in feces in cases of occult hemorrhages 297
picture, of latent tuberculosis in children and its prognostic significance..... 514
plasma, surface tension in children..... *18
platelets in newly born and very young infants 303
pressure, high, blood peptide nitrogen in. 500
pressure, treatment of hypertension.... 684
red cell sedimentation test in children.... 665
serum, calcium and phosphorus content of, in infants.....1112
study of coagulation; chemical reaction underlying proccess 126
study of connection between chlorine content of blood, blood plasma and gastric secretion 673
sugar, determination, in children by method of Weiss and Reist..... 897
sugar, glycemia in diphtheria.....1119
transfusion, amount of blood for..... 516
transfusion, fate and signifiance of donor's erythocytes in body of recipient 899
transfusion, in infancy and childhood... 158
transfusions, in severe pyuria..... 695
transfusion, syphilis transmitted by..... 313
Bone: See also osteomyelitis
development in diabetic children..... 142
effect of bone marrow extracts on blood regeneration 668
growth, physiology of..... 664
marrow, granulocytic aplasia of, following use of arsenic.....*1032
role of thyroid and parathyroid glands in chemical differentiation of bone during growth 126
transverse lines in x-ray plates of long bones of children..... 716
Book Reviews:
Autobiography of Otto Huebner..... 748
Avitaminosen und Verwandte Krankheitszustaende; W. Stepp and P. Gyoergy 747
Beitrage Zur Kenntnis der Mongoloiden Missbildung (Mongolismus and Mongoloide Idiotie); W. M. Van der Scheer 1151
Cultivating the Child's Appetite; C. A. Aldrich 949
Der Kraftwechsel Des Kindes; E. Helmreich 747
Des Kindes, Ernährung, Ernährungsstorungen und Ernährungs Therapie; A. Czerny and A. Keller.....1151
Diagnostic Therapie Der Kinderkrankheiten; F. Lust 352
Die Bronchiektasien im Kindesalter; O. Wiese 948
Keeping the baby well; J. H. West..... 166
Klinische und Serologische Beitrage Zum Scharlachproblem; G. Fanconi.....1151

Book Reviews—Continued PAGE
La Tetania Nell'Infanzia; S. Fabris.... 552
Le Syndrome Malin; V. Hutinel..... 1152
Pathological Physiology of Internal Diseases; A. W. Hewlett..... 748
Poliomyelitis; W. R. MacAusland..... 746
Potassium and Tartrates; R. W. Webster 352
Precis de Medecine Infantile. E. Weil and G. Mouriquand 1152
Systemic and Focal Immunization through Irradiation; S. Peskind..... 950
The Polynuclear Count; W. E. Cooke and E. Ponder..... 949
Tobacco and Physical Efficiency; P. Schrumpf-Pierron 166
Understanding Human Nature; A. Adler 948
University of Iowa Studies..... 748
Botelho Reaction, in diagonsis of cancer.... 497
Boyd, J. D.: Growth studies of children with diabetes mellitus.....*753
Boyden, R. E.: Influence of feeding on certain acids in feces of infants; comparison of effects of breast milk and modified cow's milk on excretion of volatile acids *580
Brain, abscess, in infants under 12 months of age; report of 2 cases with 17 collected cases *256
acute degeneration after measles.....514
aplasia of severeal nuclei of nerves of....1134
cephalocele intranasalis..... 706
cerebromeningeal lesions of new born, dependent on labor..... 138
hemorrhage, intercranial, in new born infants *443
hypoplasia pontoneocerebellaris..... 149
papilloma of the choroid plexus..... 325
Breast, procedure of De Buys as prophylactic treatment against some disorders of breast feeding..... 501
Bronchiectasis and lung cavities, diagnosis of, by intratracheal injections of lipiodol 690
types of, in children.....1125
Bronchitis, in infants..... 692
Bronchopneumonia, dextrose treatment in infants 522
Bronchoscopy, treatment of lung abscess.. 527
value of oxygen following..... 684
Bronchus, foreign body in, of child with congenital absence of other lung..... 733
Brush, N. H.: Pseudohermaphrodism (female type predominating).....*862
Buckstein, J.: Cardiospasm in infancy and in childhood: report of case in a child 11 years of age.....*427
Burns, treatment by tannic acid....546, 1137

Caffey, J. P.: Endemic meningococcus meningitis: clinical manifestations in infancy and in early childhood.....*357
Calcium, effect of, in children..... 339
metabolism, in undernourished children.*856
Cancer, Botelho reaction in diagnosis of.. 497
Capper, A.: Fate and development of the immature and of the premature child; clinical study; review of literature and study of cerebral hemorrhage in new born infant.....*263, *443
Carbohydrate metabolism, disturbance in. 133

PAGE
Celiac disease.....*414, 944
banana diet in treatment of..... 337
complicated by purpura..... 684
Cereal food, their advantages and dangers 338
Cerebellum, cerebellar tumors occurring in identical twins.....1130
Cerebrospinal fluid, fate of substances injected into the..... 327
in children.....1132
method for determining the sugar in.. 525
sugar content of..... 526
Chest, congenital defects of muscles of.. 673
funnel, treatment of patients with..... 157
Chickenpox 1067
bullous impetigo complicating varicella. 508
comparative study of pigeon pox and.... 129
development of varicella after injections of serum for whooping cough..... 156
in Sanatoria 507
nervous complications of varicola, vaccina and varicella..... 304
nuclear inclusions produced by varicella virus in testicles of monkeys..... 304
Child Welfare, health demonstration..... 739
health education in public schools..... 532
our obligation to children of our communities 532
our bureau of social welfare..... 532
Chloroma; two cases.....1147
Cholesterol and lipoid phosphorus, in infancy and in childhood; normal incidence in cord blood and during infancy *193
Cholecystography, in children..... 521
Cholelithiasis, and splenomegalic cirrhosis of the liver occurring in family..... 522
Chondrodystrophia, fetalis..... 504
Chorea, acute..... 346
minor, Czerny's phenomenon examined roentgenologically in..... 704
minor, thyrotoxic symptom complex in..1134
movements involuntary; case..... 543
treatment with injections..... 327
Chromoneuroscopy, application of, according to method of Flateau.....1132
Chylangioma, of mesentery in child.....*636
Clark, E. B.: The surface tension of blood plasma in children..... *18
Cleft Palate and hairlip.....741, 943
congenital, operative treatment..... 712
surgical correction of cleft lip and..... 530
Cod Liver Oil, antirachitic principle of... 672
concentrate in rickets and other nutritive disturbances 500
requirement curve..... 729
Cohen, P.: Effect of thyroid therapy on children *203
Cohn, D. J.: Cholesterol and lipoid phosphorus in infancy and in childhood; normal incidence in cord blood and during infancy and early childhood..*193
Colon, Hirsprung's disease 4 years after operation 739
subacute intestinal obstruction with unusual dilatation of..... 143
Communicable disease, in hospitals.....*1056
seasonal distribution.....*1051

Communicable Disease—Continued PAGE
situation in Chicago; survey.....*1048
with reference to concentration of population *1058
Conduct, disorders in children..... 146
Contractures, congenital attitude anomalies caused by muscular contractions: club-hand, wryneck and knee contractures 503
Convulsions, following insulin..... 146
Cooke, J. V.: Scarlet fever; course of development of immunity in scarlet fever *983
scarlet fever; development of toxin sensitivity of skin in infants and its relation to presence of antitoxin in blood *762
scarlet fever; relation between loss of skin sensitivity to toxin and development of antitoxic immunity during artificial immunization.....*974
scarlet fever; amphylactic factor in scarlet fever.....*991
Cooke, W. E.: The pharyngeal tonsil....*229
Coxa Vara: See Hip
Cranium, intercranial tumors of childhood.1143
Cyst, of lung, congenital.....*1024

Deaf-Mutism, after scarlet fever meningitis 677
method of teaching deaf mutes..... 154
the deaf and dumb child..... 148
Deafness, a new sound for hearing in new born: the conditioned reflex..... *36
bilateral, following poliomyelitis.....1135
Death rate, higher mortality of illegitimate infants in Berlin..... 719
infant mortality in India; is there need for an inquiry into the causes of?.. 340
infant mortality, problem of early..... 160
De Buys procedure: See Breast
Degeneration, hereditary systemic, movements involuntary; case..... 543
Dehydration, effects of, on acid-base balance of plasm of infants with mastoiditis *557
fever 318
Dermatomyositis, report of a case with necropsy *71
Dermographia, white dermographism in children 709
Dextrose, injections of, in diphtheria.... 722
treatment for bronchopneumonia in infancy 522
Diabetes 689
bone development in diabetic children.. 142
in children..... 696
mellitus, growth studies of children with.*753
mellitus, in children, course and prognosis 689
mellitus, insulin treatment of, in children 318
mellitus, synthalin in..... 693
multiple juvenile..... 945
resistant to insulin; case.....1128
Diaphragm, congenital defect of..... 504
congenital occlusion of duodenum by a complete *109
diaphragmatic hernia..... 301
eventration, congenital nonrotation of stomach with.....*641

PAGE
Diarrhea, chronic..... 736
effects of, on acid-base balance of plasma of infants with mastoiditis.....*557
Diathermy, and quartz light: their practical use..... 338
Dicephaly 502
Dick Test: See also under Scarlet Fever 140
in Sanitoria..... 507
reaction in childhood..... 678
Dick, G. F.: Etiology of an epidemic of enteritis associated with mastoiditis in infants *955
Dick, G. H.: Etiology of an epidemic of enteritis associated with mastoiditis in infants *955
Diet, banana, use of, in intestinal indigestion in children..... 337
effect of meat diet on respiratory metabolism after different types of..... 671
ketogenic 1148
ketogenic, in epilepsy.....1147
mineral, mineral metabolism on a high.*576
self selected, for newly weaned infants 744
starvation diet versus feeding in treatment of summer diarrhea..... 337
Digestive Tract, anatomy of digestion.... 298
clinical value of azotemia in digestive disturbances of infant..... 298
clinical value of phenomenon of acidosis in digestive disturbances of the infant 299
dehydration and the choleriform syndrome 299
physiology of digestion..... 672
study of digestive disturbances of the infant 298
Diphtheria *1069, *1091
association of measles and..... 509
antitoxin production..... 508
bacillus, coccal forms of the..... 497
carriers 743
combined active and passive immunization against.....1120
differential diagnosis of..... 309
diphtheritic intoxication and muscular lesions 510
glycemia in.....1119
heart block in..... 731
in Sanitoria..... 507
injections of dextrose in..... 722
intradermal test for the virulence of diphtheria cultures..... 508
negative phase in inoculations..... 510
prevalence of, in the United States....1118
preventative measures in.....1118
problems in..... 128
pulmonary gangrene, following: report of case.....*250
short survey of prevention work in Edinburgh 307
the heart after severe..... 506
toxin in dogs, metabolism following injection of..... 665
toxins producing a diptheroid bacillus.. 128
Diseases of children in 1927; annual review 717
Diuretics, effects on infants..... 340
Diverticulum, congenital, of urinary bladder 136

Diverticulum—Continued PAGE
ileum, with patent ductus arteriosus, hematoma of left suprarenal gland, meningeal hemorrhages: report of case in a Chinese infant....*254
Meckel's 1149
Meckel's, perforated gangrenous, in a new born infant....*438
Dodge, E. F.: Antenatal infection a factor in fetal morbidity....*221
Duodenum, congenital occlusion of, by complete diaphragm....*109
pyloric obstruction in an infant from pressure of a distended gall bladder 320
ulcers of, with faulty development of pancreas in an infant 7 weeks old.. 322
Duskes, E.: Antenatal infection a factor in fetal morbidity....*221
Dysentery, amebic....*1051, *1073
amebic, with motion picture demonstration of amebas.... 541
and appendicostomy.... 335
citric acid milk as diet for dysenteric children 1114
bacteriologic study of, in Porto Rico..1120
(Flexner's) with multiple peripheral paralysis 541
peculiarities of, in infancy.... 309
Dysostosis cleidocranial.... 735
Dystonia musculorum, movements involuntary; case.... 543
Dystrophy, adiposogenital, of hypophysial cerebral origin.... 322
in artificially fed infants....1113
interrenal; case.... 521
maxillary facial dystrophies....1126

Eczema, and impetigo, most frequently seen dermatologic conditions in infants and young children.... 163
clinic for the study of asthma, eczema and 125
in infants and young children.... 528
infantile 738
infantile, relief of itching by epinephrine 155
Edema 945
Eley, R. C.: Scarlet fever antitoxin.... *14
Embryology, injury to embryo and syphilis 1124
internal secretions affecting growth of embryo 134
Emphysema, mediastinal in new born.... 674
study of human pleura pulmonalis; its relation to blebs and bullae of.... 316
Empyema, autotherapy of.... 696
treatment in children; method.... 530
Encephalitis *1075
after vaccination.... 679
comparison of virus obtained by Kobayashi from epidemic.... 666
due to sulpharsphenamine poisoning.... 313
epidemic, acute.... 697
epidemic, and paralysis agitans, sugar metabolism in.... 148
hereditary influences in postencephalitic psychic disturbances.... 147
meningo, and toxic meningitis, due to ascaris infestation.... 327

Encephalitis—Continued PAGE
postvaccinalis, problem of the "pure" smallpox vaccine in connection with. 310
rare sequela of epidemic....1132
Encephalocele, anatomic study and surgical treatment in four cases.... 335
Encephalomyelitis 697
complications of variola, vaccina and varicella 305
Endocarditis, lenta, in childhood.... 694
subacute bacterial.... 725
subacute infective, in congenital morbus cordis 519
Endocrine glands, fats and lipoids in endocrine organs.... 669
in relation to infancy and childhood....*872
Endothelioma, in an infant.... 332
Enteritis, etiology of epidemic of, associated with mastoiditis in infants....*955
Eosinophilia, case report.... 347
Ephedrine, clinical use of.... 338
Epidermolysis, bullosa hereditaria.... 727
Epilepsy, attempts at producing epileptic and hysterical attacks by deep breathing 527
etiology 946
in children....*653
in childhood.... 147
influence of ketogenic diet on....1134, 1147
subcortical 701
treatment in childhood.... 147
with myoclonus.... 526
Epinephrine, effects of intracutaneous injection of.... 687
effect of, on blood picture in normal, lymphatic and scrofulous children.... 159
for relief of itching in infantile eczema 155
Epiphyses, unilateral hydrarthus with bilateral gummatous epiphysitis of tibiae and psychic infantilism....1125
Ergosterol, and antirachitic vitamin D, literature regarding 899
Erysipelas 944, *1071
in infants, treatment.... 540
in new born.... 947
treatment with mercurochrome-220 soluble and polyvalent antistreptococcus serum 164
Erythema, centrifugal annular.... 157
nodusum, and rheumatic conditions.... 529
nodusum, incubation time.... 709
Erythrocytes, diameters, study of, in newborn 505
sedimentation rate of, in infantile tuberculosis 513
Erythrodermia, edematous vesicular, cured by excision of intragluteal nodules of bismuth 156
Leiner's desquamative, treatment of, with injection of mother's blood.... 333
Esophagus, esophageal stenosis.... 692
papilloma of; report of case.... 536
unusual complications after injuries to, by foreign bodies and other ways: comments on roentgen-ray diagnosis.. 323
Exostoses, diaphysical aclasia.... 691
Eye, congenital cataract.... 328
thrush of conjunctiva.... 328

PAGE

Falk, I. S.: Survey of the communicable disease situation in Chicago.....*1048
Fatigue, in children..... 717
Fats and lipoids in certain endocrine organs 669
Feces, influence of feeding on certain acids in feces of infants.....*580
nature of the derivatives of the blood pigment found in the feces in cases of occult hemorrhages..... 297
Feldstein, G. J.: A breast and bottle shy infant; nursing only when asleep....*103
Femur, congenital absence of..... 546
Fetus, cause of fetal death in 144 cases; child life investigations..... 905
infection antenatal, a factor in fetal morbidity *221
Fever, due to eating, thirst and crying (?) 664
in children, external heat cause of..... 158
salt, subcutaneous.....1113
Fibrocystic disease of humerus.....1138
Folliculitis ulerythematosa reticula combined with lichen spinulosus, epiderma cysts and folliculitis decalvans..... 707
Fontanel, occlusion of, in normal and diseased states.....1111
Food, irradiated, experience with, after ether extraction.....1115
Foot, flatfoot in children..... 342
Fracture, complicated, of forearm.....1138
Fragilitas ossium, its hereditary aspect; case 687
Frank, M.: Metabolism of undernourished children; calcium metabolism.....*856
Friedman, L. S.: The urinary leukocyte count in children in normal and in pathologic conditions.....*200
Froin's Syndrome: See Xanthochromia
Furunculosis in infants..... 529

Gallbladder, pyloric obstruction in an infant from pressure of a distended..... 320
Gangrene, of leg and arm..... 691
pulmonary, following diphtheria; report of case.....*250
Ganglioneuroma, transformation of a malignant paravertebral sympathicoblastoma into a benign ganglioneuroma..... 317
Garvin, J. A.: Congenital occlusion of duodenum by a complete diaphragm.*109
Gaucher's Disease: See Splenic Anemia
Geiger, J. C.: Survey of the communicable disease situation in Chicago....*1048
Gerstley, J. R.: Influence of feeding on certain acids in feces of infants; comparison of effects of breast milk and modified cow's milk on excretion of volatile acids.....*580
Survey of the communicable disease situation in Chicago.....*1048
Giardiasis: See Lambliasis
Gliosis: See Neuroglia
Goiter, exophthalmic..... 737
simple, heredity in..... 519
Gonorrhea, fate of children infected with 510
Gordon, M. B.: Cholesterol and lipoid phosphorus in infancy and childhood: normal incidence in cord blood and during infancy and early childhood..*193

PAGE

Gottschall, G.: Respiratory metabolism in infancy and in childhood; respiratory exchange in marasmus; basal metabolism *615
Gower, C.: Growth standards of height and weight for girls in private schools.*411
Gray, H.: Growth standards of height and weight for girls in private schools...*411
Greene, D.: Effect on urine of addition of acids and alkalis to diet of infants.... *38
Greengard, J.: Pericarditis as a primary clinical manifestation of tuberculosis in childhood.....*631
Greenwald, H. M.: Congenital nonrotation of stomach with eventration.....*640
Growth and development of immature and premature child *262, *443
body, action of various extracts..... 897
physical, of children..... 741
standards of height and weight for girls in private schools.....*411
studies of children with diabetes mellitus *753
stunted 518
Grulee, C. G.: Congenital Syphilis; treatment by intraperitoneal injection of necarsphenamine: report of 2 years work *47
Survey of the communicable disease situation in Chicago.....*1048
Gymnastics and massage of infants in ancient Rome.....1141

Hair Lip and cleft palate..... 741
Hand, Madelung's disease of hand; etiology 1139
measurement of strength of, and of vital capacity of lungs, in children and young adults..... 296
Hartmann, A. F.: Chemical changes in body as result of certain diseases; effects of diarrhea, vomiting, dehydration and oliguria on acid base balance of plasma of infants with mastoiditis.*557
Hassin, G. B.: Amaurotic family idiocy (Tay-Sachs type): case with protracted course *87
Hawks, J. E.: Metabolism of undernourished children; protein metabolism..*968
Hauver, R. B.: Intradermal vaccination.*186
Hay-fever, clinic for study of asthma, eczema and..... 125
Hays, B. B.: Metabolism of undernourished children; protein metabolism...*968
Hearing: See Deafness
Heart: See also Arrythmia
after severe diphtheria..... 506
bicameral dextrocardia with systemic pulmonary circulation 741
block, congenital..... 724
block, functional partial.....1129
block in diphtheria with recovery..... 731
cardiac failure in children under 5 years of age; with special reference to acute rheumatic myocarditis..... *1
diagnosis and treatment of cardiac conditions 688
disease, rheumatic 317
disease, urobilinuria in children with... 535
disease, rheumatic 317, 684
displacement, in children with pneumonia 516

Heart—Continued PAGE
effect of insulin on diseased myocardium. 1130
electrocardiographic records in hearts of normal children. *1001
functional cardiac disorders; symptoms, signs and treatment. 319
mitral disease, differential diagnosis of early pulmonary tuberculosis and. 516
of normal children, studies. *791
of normal children; clinical studies including notes on effort syndrome. *398
pure congenital dextrocardia. 144
transposition of aorta and pulmonary artery. 136
Height, and weight, growth standards of, for girls in private schools. *411
Hematoma, left suprarenal gland, ductus, arteriosus, meningeal hemorrhages and diverticulum of the ileum: report of case in Chinese infant. *254
Hemihypertrophy and twinning. 133
Hemiplegia, significance of flexor posture of upper limb, with an account of a quadrupedal extensor reflex. 700
Hemophilia, Kirchheimer families of bleeders. 321
Hemoptysis, fatal hemoptysis in childhood 521
Hemorrhage, in suprarenals, in case of status lymphaticus. 543
intra-abdominal in boy of six years. 1125
intracranial, in new-born infants. *262, *443
into lungs of new-born infants. 302
diaphragmatic. 301
diaphragmatic, rupture of hernia sac. 694
indications for operation in infant and young child. 335
Hexylresorcinol, bactericidal activity of, in glycerin. 336
Hip, congenital dislocation of. 157
traumatic coxa vara. 157
Hirschprung's Disease: See Colon
Hodgkin's Disease: See Lymphogranuloma
Homatropin, influence of acidity on activity of atropine and. 340
Hoyne, A. L.: Prevention and modification of measles with antimeasles diplococcus goat serum. *1021
Humerus, myeloma of. 691
Hunger, central factors in. 665
Hunter, W. C.: Perforated gangrenous Meckel's diverticulum in new-born infant: report of case. *438
Hydrocephalus. 740
unusual form of. 523
Hydrotherapy, experiments on therapeutic value of hot baths with special reference to treatment of syphilis and some physiologic observations. 312
Hypercholesterolemia, and Xanthomatosis cutis. 332
Hypertension: See Blood Pressure
Hypertrophy, congenital. 726
congenital, involving right side of child 673
of infancy and childhood. *885
Hypospadius, in male, operative treatment of. 714
Hysteria, attempts at producing epileptic and hysterical attacks by deep breathing. 527

Icterus: See Jaundice PAGE
Idiocy, amaurotic family, (Tay-Sachs type); case with protracted course. *87
mongolian, skull of mongolian imbecile. 523
pathology of the cerebral cortex in. 524
Ileum, diverticulum, with patent ductus arteriosus, hematoma of the left suprarenal gland, meningeal hemorrhages: report of a case in a Chinese infant. *254
Immature infants, fate and development of premature and; clinical study. *262, *443
Immunity against scarlet fever. *974
combined active and passive, against diphtheria. 1120
course of development of, in scarlet fever. *983
experimental, in poliomyelitis. 666
Impetigo, bullous, complicating varicella. 508
and eczema, most frequently seen dermatologic conditions in infants and young children. 163
Infants' feeding. 502
breast and bottle shy infant; nursing only when asleep. *103
effect on urine of addition of acids and alkalis to the diet of infants. *38
in relation to diseases at all ages. 500
influence of, certain acids in feces of infants. *581
interesting comparisons in feeding schedules for full term new-born infants. 301
persistent vomiting in breast fed infants 501
problems in private practice. 298
Infant hygiene, present status of maternal and infant hygiene in the United States. 716
Infantilism, generalized. 697
Infantilism, psychic, and unilateral hydrarthrus with bilateral gummatous epiphysitis. 1125
Infant Welfare, and protection of motherhood in India. 1140
instructions for young girls. 159
laws pertaining to education of teachers for. 159
Infection, antenatal, factor in fetal morbidity. *221
blastomycotic, treatment of by gentian violet. 1112
effect of exposure to the sun on susceptibility of rachitic rats to. 295
focal, relationship of, to general disease in infants and young children. 716
infectious diseases, etiology, prophylaxis and serum therapy of. 1121
joint and bone, in children. 334
rheumatic, and rheumatism nodules. *823
roentgen treatment of spontaneous, posttraumatic and postoperative coccus infections and suppurations. 336
Influenza. *1069
enlargement of lymph nodes in. 510
infantile. 510
lymphatic reaction in. 1128
Injuries, birth, the unborn babe and. 134
Institutional care of children, organization and method. 721
Insulin, convulsions following. 146
diabetic child resistant to. 1128

Insulin—Continued PAGE
effect of, on diseased myocardium.....1130
treatment of diabetes mellitus in children 318
Intelligence, some prognostic values in measurement of..... 699
Intestines, acute intestinal obstruction.... 944
congenital encapsulation of the small intestine 503
lymphatic tumor of, causing obstructions 742
perforation of, in baby..... 691
sarcoma of ileum with partial obstruction 742
Intussuception 946
Intussusception, acute, manipulative reduction under fluoroscopic control..... *61
Iodized oil, injections transglottically in children 153
use in roentgen-ray diagnosis of spinal lesions 522

Jaundice, acholuric family..... 518
congenital hemolytic icterus.....165, 1150
hereditary hemolytic, complicated with acquired icterus..... 522
icterus neonatorum and water economy. 675
icterus neonatorum of calves..... 505
report of investigation into outbreak of infectious jaundice..... 308

Kaucher, M.: Metabolism of undernourished children; calcium metabolism....*856
Kaufman, J.: Congenital nonrotation of stomach with eventration.....*640
Keloid, roentgen-ray treatment of..... 528
Kidney, functional capacity of, and its importance in practice.....1126
malformation of..... 502
nephrosis, 3 cases..... 343
surgical results of tumors of, in childhood 713
urogenital diseases in infancy.....1129
Koch, L. A.: Dermatomyositis: report of a case with necropsy..... *71
Krahulik, L.: Roentgenographic changes in thorax of normal new-born babies; daily roentgenographic study of 25 normal babies during the first 14 days of life *837

Lambliasis, giardiasis in an infant..... 144
lamblia intestinalis, case..... 165
Larynx, microscopic changes in larynx in whooping cough.....1121
Leonard, B. W.: Cardiac failure in children under 5 years of age, with special reference to acute rheumatic myocarditis *1
Leukemia, lymphatic..... 944
lymphatic, neoplastic nature of, and its relation to lymphosarcoma..... 517
Leukemia myelogenous..... 724
Leukocyte, count, urinary, in normal and pathologic conditions in children....*200
variations of, in new-born after eating.. 505
Levine, S. Z.: Respiratory metabolism in infancy and in childhood; elimination of water through the skin and respiratory passages in infants..... *54
Respiratory metabolism in infancy and in childhood; respiratory exchange in marasmus; basal metabolism.....*615

PAGE
Lichen planus atrophicus? of zosteriform distribution in a child aged 3 years.. 156
planus, in infancy..... 708
planus striatus of Radcliffe Crocker.... 708
Lincoln, E. M.: Studies on the hearts of normal children; roentgen-ray studies.*791
The hearts of normal children; electrocardiographic records.....*1001
The hearts of normal children; studies, including notes on effort syndrome..*398
Lipoids, phosphorus, and cholesterol in infancy and childhood; normal incidence in cord blood and during infancy and early childhood.....*193
Liver, acute liver insufficiency in childhood 696
cirrhosis of, in infancy; cases..... 735
cystic pathogenesis of..... 135
function test..... 514
hypertrophic cirrhosis of, in infancy and childhood 687
injury of at birth, giving the clinical picture of congenital absence of the bile ducts..... 301
splenomegalic cirrhosis of liver and cholelithiasis occurring in a family.. 522
Lung, abscess, from the medical standpoint 316
abscess, roentgen-ray diagnosis of..... 317
acute massive atelectasis of.....*239
cavities, diagnosis of, by intratracheal injections of lipiodol..... 690
congenital cyst of.....*1024
differential diagnosis of tuberculous and nontuberculous pulmonary disease in childhood 1124
foreign body in bronchus of child with congenital absence of other lung..... 732
forensic importance of microscopic examination of lungs in infants dying at or about the time of birth..... 302
gangrene of, following diphtheria; report of case.....*250
measurement of vital capacity of, and of strength of the hand, in children and young adults..... 296
Lupus erythematosus; action of bismuth in 156
Lymphatism, mechanism of death in status lymphaticus 688
status thymicolymphaticus considered in light of recent work on thymus..... 686
Lymphogranuloma; case..... 521
(Hodgkin's disease) of the sternum..... 518
placental transmission of Hodgkin's disease 136
Lymph Nodes, abdominal, tuberculosis of, in early life..... 511
enlargement of, in grip..... 510
radium therapy in tuberculous cervical adenitis 511
Lymphosarcoma, neoplastic nature of lymphatic leukemia and its relation to 517

Madelung's Disease: See Hand
Malaria, for treatment of congenital taboparesis 522
malarial amaurosis; case.....1135
Malformations, early embryonic causes, in the nervous system..... 137
Manning, J. B.: Pseudohermaphrodism (female type predominating).....*862

PAGE

Marasmus, the respiratory exchange; basal metabolism *615
Mastitis, infecting organisms in four dairy herds 130
Mastoids, apparatus for stereoscopic roentgenography of..... 151
is mastoidectomy necessary in young children? 330
process, bilateral symmetrical breaking down of.....1136
Mastoiditis, effects of diarrhea, vomiting dehydration and oliguria on acid-base balance of plasma of infants with....*557
etiology of epidemic of enteritis associated with.....*955
infantile, with gastro-intestinal symptoms 705
pathology of, in infants..... 706
McLean, S.: Endemic meningococcus meningitis: clinical manifestations in infancy and in early childhood.....*357
Measles 508
acute degeneration of brain after measles; report of 2 cases..... 514
antimeasles diplococcus serum..... 507
association of diphtheria and..... 509
complication following 509
earlier diagnosis of..... 163
German *1071
in Sanatoria..... 507
inquiry into relationship between housing conditions and incidence and fatality of 1119
preventative measures in.....1118
prevention and modification of, with antimeasles diplococcus goat serum..*1021
prophylaxis of, with suggested scheme in dealing with epidemics..... 307
second illness in..... 140
Meinicke reaction..... 681
Meninges, permeability, experimental study in inflammatory conditions..... 524
staphylococcus infection of spinal.....1131
Meningitis, case of colimeningitis..... 526
cerebrospinal *1075
due to Pfeiffer bacillus..... 326
endemic meningococcus; clinical manifestations in infancy and early childhood.*357
epidemic meningococcus..... 147
in mumps..... 309
meningeal permeability in childhood.... 704
meningoradiculitis 326
serosa, critical remarks on..... 154
spirochetal purulent..... 703
staphylococcus infection of spinal meninges 1131
syphilitic, treatment with tryparsamide in 312
toxic, and meningo-encephalitis due to ascaris infestation..... 327
treatment of purulent meningitis from the pediatrician's standpoint.....1133
tuberculous, etiology of secondary.....1122
uranin as aid in diagnosis of.....1134
Mental hygiene movement, has the pediatrician any place in?.....1131
Merritt, K.: Rheumatic nodules, their incidence in rheumatic infections....*823
Merselis, J. G.: Pulmonary gangrene following diphtheria: report of case....*250
Mesentery, chylangioma of, in child.....*636

PAGE

Messeloff, C. R.: Cardiospasm in infancy and in childhood.....*427
Metabolism; See also under Calcium; Carbohydrate
at puberty..... 300
basal, respiratory exchange in marasmus.*615
following injection of diphtheria toxin in dogs 665
influence of parenteral infections and intoxication on metabolism of chlorine, calcium and magnesium..... 903
investigations in adipose children at puberty 300
investigations in protein metabolism in fever by means of the peptidase reaction 296
mineral, on high mineral diet.....*576
mineral, effect of parathyroid extract on, in infantile tetany.....*392
of undernourished children,.....*856, *968
peculiarities of growing organism..... 898
respiratory, in infancy and in childhood *54
sugar, in epidemic encephalitis and paralysis agitans..... 148
Metaldehyde poisoning; case..... 533
Methyl salicylate, poisoning..... 716
fatal poisoning; case..... 532
Microcephaly, and diffuse gliosis..... 701
Microsomia, essential..... 692
Middleton, R. P.: Auricular fibrillation in childhood: report of cases..... 420
Milk, acidified, use as a permanent diet.. 900
analysis of reindeer milk and its use as a food in infancy..... 133
antirachitic activation of, its relation to content of stearin.....1115
breast, concentrated, for premature infants 737
citric acid, as diet for dysenteric children 1114
concentrated, public health value of.... 500
difficulties of judging inability to nurse 132
excretion of antiseptic dyes through the mammary gland..... 130
growth and feeding of infants; pathogenesis of dystrophy from cow's milk 131
increase of soluble nitrogen in infant's stomach 299
indications for a milk free diet..... 132
irradiated, alleged scorbutigenic effect of 904
lactic acid, and lemon juice milks; clinical experience with..... 901
management of breast milk stations.... 717
secretion, action of atropine on..... 298
types of hemolytic streptococci in certified 130
Mitchell, A. G.: The urinary leukocyte count in children in normal and in pathologic conditions.....*200
Mononucleosis, infectious..... 549
Moore, C.: Dermatomyositis: report of a case with necropsy..... *71
Mortality: See Death Rate
Movements, involuntary; 4 cases..... 543
Mumps: See under parotitis
Muscles, of chest, congenital defects of.. 673
Myatonia, congenital.....1133
congenita, Oppenheim; report of unique case *82

PAGE

Myeloma of humerus..... 691
Myocarditis, rheumatic, cardiac failure in children under 5 years of age with special reference to..... *1
Myositis, ossificans progressiva.....143, 692

Nasopharynx, importance of nasopharyngitis 528
Nelson, M. V.: Growth studies of children with diabetes mellitus.....*753
Nephritis, acute, study of inorganic constituents of serum in children with.. 685
(chronic, glomerular) 548
(parenchymatous); nephrosis, 2 cases.. 548
sugar treatment in.....1127
tubular, in children and its relation to other forms of nephritis..... 685
Nephrosis: See under kidney
Nerves, aplasia of several nuclei of nerves of brain.....1134
paralysis of right diaphragm in new-born due to phrenic nerve injury.... 138
Nervous system, early embryonic causes of malformations in..... 137
neurologic diagnosis in children.....1142
nervous symptoms provoked by intestinal worms 1127
operations on sacral sympathetic nerve in infants 1138
vaccination disturbances of..... 149
Neuritis, multiple..... 550
retrobulbar 542
Neuroblastoma, of the suprarenal in young children 163
sympathetic 295
sympathetic, transformation of malignant paravertebral sympathicoblastoma into a benign ganglioneuroma..... 317
Neuroglia, microcephaly and diffuse gliosis 701
periodic decrease in abilities of talented children 148
Neurosis, of vegative system, in small child 703
Neurosurgery, in new-born and children.. 711
Neurosyphilis, treatment of..... 312
Nevi, nevus pigmentosus pilosua..... 728
pigmentary, of human skin.....1136
vascular, congenital..... 946
New-Born infants, hemorrhage, intracranial in *443
roentgenographic changes in thorax; study of 25 normal babies during first 14 days of life..... 837
variation of leukocytes in, after eating.. 505
Nicolson, G. H. B.: The hearts of normal children; electrocardiographic records.*1001
Nielson, J. M.: Myatonia congenita of Oppenheim; report of a unique case.. *82
Niemann-Pick's disease.....1126
Norton, J. F.: Survey of the communicable disease situation in Chicago..*1048
Nursing Infants, operability of..... 718
Nutrition: See also Athrepsia
etiology of atrophic conditions in children and the role of protoplasmic dispersity in diagnosis..... 133
growth and feeding of infants; pathogenesis of dystrophy from cow's milk 131

Nutrition—Continued PAGE
influence of cooking on nutrition of food 502
of child in first 2 weeks of life, within and without closed institutions..... 131
predisposition and..... 501
the malnourished child..... 337
Nystagmus, and spasmus nutans..... 704

Olcott, C. T.: Pulmonary gangrene following diphtheria: report of case....*250
Oleothorax 690
Onychoptosis, recurrent, occurring as a family disorder 332
Oppenheim's Disease: See Myatonia Congenital
Osteochondritis deformans juvenilis (Perthes' disease) histopathogenesis of 669
Osteomyelitis, acute, of superior maxilla in young infants..... *65
acute, of superior maxilla with large infra-orbital abscess in a nursing infant 330
acute rapidly fatal.....1127
of cervical spine with cerebrospinal symptoms 336
of long bones in infant..... 314
Osteosarcoma, of chest wall, in girl of 11 years 1138
Osteosclerosis fragilis, (Albers-Schonberg's disease); case..... 320
Otitis Media, acute, in infants..... 151
in infants..... 329
Oxycephaly, case..... 301
Oxygen, value of, following bronchoscopy. 684

Pancreas, ability of infant's pancreas to digest cell nuclei..... 672
faulty development, and ulcer of duodenum, in an infant 7 weeks old.... 322
Papilloma, of choroid plexus..... 325
of esophagus; case..... 536
Paracentesis tympani, a practitioners operation 153
Parietal bones, abnormally large foramina parietalia 673
familial partial defect 741
Paralysis, agitans, and encephalitis epidemic, sugar metabolism in..... 148
fatal acute ascending, following the use of antirabies vaccine in child bitten by rabid dog 703
histologic observations in 2 cases of progressive 151
infantile, after care of..... 702
infantile, fund for study of..... 663
infantile, muscle training in..... 699
infantile, treatment by Rosenow's serum in 325
juvenile, its salient characteristics with special reference to infantilism..... 311
right diaphragm, in new-born due to phrenic nerve injury..... 138
Parathyroid, extract, effect of on mineral metabolism in infantile tetany.....*392
role of thyroid and parathyroid glands in chemical differentiation of bone during growth 126
spasmophilia with hypoplasia of thymus and 526
Paratyphoid in infants; cases.....1120

PAGE

Parkinsonism, postencephalitis, movements involuntary; case....543
Parmelee, A. H.: Amaurotic family idiocy (Tay-Sachs type): a case with a protracted course *87
Parotid gland, recurrent swelling of.... 695
Parotitis, in Sanatoria.... 507
meningitis in.... 309
ocular complications in.... 139
Patrick, H. T.: Epilepsy in children....*653
Peacock, S.: Prevention and modification of measles with antimeasles diplococcus goat serum....*1021
Pellagra in childhood....1137
Pemphigus, in child.... 331
Peptic Ulcer, duodenum, with faulty development of pancreas in infant 7 weeks old 322
gastric rupture of, in infancy.... 133
Peptidase, investigations in protein metabolism in fever by means of peptidase reaction 296
Pericarditis, primary clinical manifestation of tuberculosis in childhood....*631
Periostitis hypertrophic, juvenile.... 694
of fingers and toes 743
Peritonitis, chronic, in childhood.... 695
omphalic 521
tuberculous 547
Perthes' Disease: See Osteochondritis deformans juvenilis
Pertussis: See Whooping Cough
Pharynx, pharyngeal tonsil....*229
Phrenicectomy, in children.... 711
Physical defects in school children, correction of 338
Physical development, of child groups, estimation of 1111
Physical exercise, effect of, on child development 1140
Photoactivity, antirachitism.... 300
Pineal gland, pathology of....1130
Pirquet reaction: See Tuberculin
Pituitary Body, contribution to anatomy and functions of hypophysis 126
body, dystrophy adiposogenital of hypophysial cerebral origin.... 322
Pigmentation, mongolian blue patches....1137
reticulated and diffuse....1137
Platou, E. S.: Congenital cyst of lung...*1024
Pneumococcus planus; etiology of influenzal diseases 669
Pneumoencephalomyelography, value of a simplified method 146
Pneumonia *1065
heart displacement in children with.... 516
lobar, in new-born infants.... 304
pathologic anatomy of pertussis with special reference to pneumonia caused by pertussis bacillus....507
Pneumothorax, artificial, in childhood.... 521
artificial, treatment of pulmonary tuberculosis 511
completion of inadequate treatment of pneumothorax in tuberculous children by endopleural interference.... 683
in child of 6 years....1125

PAGE

Poisoning: See Metaldehyde; Methyl Salicylate; Shoe Dye; Sulpharsphenamine, etc. 945, *1075
Polio-Encephalitis: See Encephalomyelitis, Poliomyelitis
anterior 702
bilateral deafness following....1135
experimental immunization in.... 666
orthopedic treatment of acute stage of anterior 539
reports 699
Precipitin reaction, observations with.... 666
Pregnancy, the unborn babe....134
Premature infants, a new sign of prematurity 136
fate and development of immature and...*443
fate and development up to early school age 905
Protein, hypersensitiveness, transmission of, from mother to offspring....1115
therapy, experimental tests....1113
therapy, treatment with preparation of nucleins and phenol in pediatrics.... 140
Pseudohermaphrodism, female type predominating *862
Psoriasis, hyperkeratotic, with papillomatosis and cutaneous horns: lingual lesions of undetermined nature.... 156
Psychology of childhood.... 704
Purpura, celiac disease complicated by.... 684
fulminans with symmetric bleedings.... 324
Pyelitis, in infancy and early childhood....1125
mode of infection in.... 347
obstruction and cause of persistent urinary infection.... 946
Pylorus, hypertrophic pyloric stenosis.... 689
operative treatment of pyloralspasm in infants 712
stenosis of infancy 531
stenosis, treatment in infants.... 531

Quarantine *1103
Quartz light, and diathermy; their practical use 338

Rabies, comparison of virus obtained from encephalitis and.... 666
in children....1120
Radium therapy in tuberculous cervical adenitis 511
Rat-Bite fever, causative organism and its use in treatment of paresis.... 540
(sodoku) 692
Reflex, eye reflex in neck in early infancy.. 703
forced grasping and groping.... 701
method of examining the conditioned food reflexes in children and in mental disorders 700
Respiratory tract, systemic manifestations of infections in upper.... 144
Rhabdomyosarcoma of vulva in children.... 157
Rheumatic fever, bacteriologic studies of a nonmethemoglobin-forming streptococcus with special reference to its soluble toxin production.... 305
skin reaction in.... 544
Rheumatism, juvenile, notes on.... 306
rheumatic nodules, their incidence in rheumatic infections *823

PAGE

Rickets 337, 670
chemistry findings in..... 498
clinical observations on cod liver oil concentrate in rickets and other nutritive disturbances 500
concerning dose of antirachitic factor in prevention and cure of rickets..... 729
direct and indirect light therapy of..... 904
effect of antirachitic vitamin on phosphorus calcium and *p*H in intestinal tract 500
etiology of 130
experimental 299
further communication on question of glycolysis 132
infrequency of severe rickets in New Orleans *590
literature regarding ergosterol and the antirachitic vitamin D..... 899
plea for sanity in the use of modern methods for prevention and treatment of 499
prevention and treatment of..... 499
rats, susceptibility to infection after exposure to the sun..... 295
renal, observations on bone changes in.. 319
treatment of 501
Ringworm, detection of ringworm hairs on scalp of their fluorescence under ultraviolet light 332
infection in horses and immunity in experimental ringworm 707
scalp, treated by thallium acetate epilation 155
therapy by thallium acetate..... 529
treatment with thallium..... 331
Robinson, S.: Pseudohermaphrodism (female type predominating).....*862
Roentgenography. Roentgenograms and photographs, from Babies' and Children's hospital; demonstration..... 720
roentgenographic changes in thorax of new born infants; study of 25 cases... 837
stereoscopic apparatus for mastoids..... 151
Roentgen-Ray, diagnosis, comments on: unusual complications after injuries to esophagus by foreign bodies and in other ways 323
diagnosis of lung abscess..... 317
treatment of keloids..... 528
treatment of spontaneous posttraumatic and postoperative coccus infections and suppurations 336
observations in asthmatic children..... 540
studies on hearts of normal children....*791
treatment of glands and bones in children 533
Rosenbaum, H. A.: Juvenile tabes dorsalis *866
Rothman, P. E.: Cardiac failure in children under 5 years of age, with special reference to acute rheumatic myocarditis *1
Royster, L. T.: Chylangioma of mesentery in a child *636
Ruhräh, J.: Bartholomaeus Metlinger; ?-1491 .....*492
Cornelius Roelans; 1450-1525..... 659
Leonellus Faventinus Victorius ?-1520; George Khufner, Jr., ?—? *1106
Sebastian Oestereicher *894
Soranus of Ephesus.....*120
Paulus Bagellardus. ?-1492.....*289
Rumination in childhood.....1127

PAGE

Sadler, W.: Congenital cyst of lung.....*1024
Sanford, H. N.: Abscess of brain in infants under 12 months of age; report of 2 cases with 17 collected cases.....*256
Congenital syphilis; treatment by intraperitoneal injection of neoarsphenamine: report of 2 years work..... *47
effect of ultraviolet light on blood of new-born infants; erythrocytes and hemoglobin *9
Sarcoma, of ileum, with partial obstruction. 742
Scarlet Fever.....509, *1069, *1087, *1092
anaphylactic factor in.....*991
antitoxin *14
avoiding shocklike effects in treatment of, with convalescent serum..... 676
course of development of immunity in...*983
criticism of Dick conception of scarlet fever based on clinical observations and results 140
development of toxin sensitivity of skin in infants and its relation to presence of antitoxin in blood.....*762
etiology of..... 308
experiences with antitoxic scarlet fever serum 678
histology of deafmutism after scarlet fever 677
in Sanitoria 507
preventative measures in..... 1118
relation between loss of skin sensitivity to toxin and development of antitoxic immunity during artificial immunization..*974
researches on 677
skin blanching by commercial antitoxins.*607
supervision stability of the blood in scarlatina 311
surgical 675
test of intradermal salt solution..... 304
toxic moiety of streptococcus scarlatine.. 675
vitality of scarlatinal streptococci..... 498
Schilling, S. J.: Vitamin requirements of nursing young; the production of berbiberi in the nursing young (mus norvegicus albinus) associated with hemorrhages *811
Sclera, blue, and multiple fractures..... 549
Scott, A. V.: Patent ductus arteriosus, hematoma of the left suprarenal gland, meningeal hemorrhages and diverticulum of the ileum: report of case in a Chinese infant *254
Scrofula, a case mistaken for Swift's disease 165
Scurvy, chemical composition of bones in.. 502
infantile, associated with dry milk..... 899
infantile, subperiosteal hemorrhage in.... 549
influence of purulent infection on development of experimental..... 127
Sedimentation test, value of, in surgical tuberculosis 679
Septicemia, agranulocytic sepsis, case of.... 165
otitic origin via the jugular vein..... 330
puerperal sepsis *1073
Serum, prevention and modification of measles with antimeasles diplococcus goat serum *1021
study of inorganic constituents of, in children with acute nephritis..... 685
Sex factor in infantile tetany.....*964
Shoe dye poisoning 542
report of 3 cases.....*1038

PAGE

Shohl, A. T.: Mineral metabolism on a high mineral diet *576
the effect of parathyroid extract on mineral metabolism in infantile tetany. *392
Shorr, E. Y.: Mineral metabolism on a high mineral diet *576
the effect of parathyroid extract on mineral metabolism in infantile tetany *392
Shulman, H. I.: Cardiospasm in infancy and in childhood; report of case in child 11 years of age *427
Sinus, nasal accessory, sinusitis in children 152
superior longitudinal, complications in puncture of 526
thrombosis, lateral, streptococcus mucus infection causing 152
Sinusitis, in children 943
Skin, cutis laxa 708
diseases in new born; 2 cases 675
diseases, review of literature on skin diseases in children in 1926 717
fungus disease of 709
reaction in childhood to different irritants 708
relations between skin and 671
tests in scarlet fever, the development of toxin sensitivity of skin in infants and its relation to presence of antitoxin in blood *762
tests tissue hydration and its relation to susceptibility and immunity as shown by skin tests in asthma chronic sinusitis and other infections 687
Skull, asymmetry in, of the new-born 303
Smallpox *1075, *1092
nervous complications of variola, vaccina and varicella 304
studies in 743
vaccination, tetanus following 699
vaccine, method for estimating the potency of 1118
Social Service, among infants and small children 159
Otto Huebner's work in 160
the child society fears 160
Societies, directory of, Canadian 353, 553, 749, 951, 1153
Societies, directory of, local 167, 354, 554, 952, 750, 1154
Societies, directory of, national 167, 353, 553, 749, 950, 1153
Society Transactions: Chicago Pediatric Society 545, 933, 1142
North Pacific Pediatric Socity 943
Northwestern Pediatric Society 341, 1149
Pediatric Section of the Academy of Medicine of Cleveland 348
Pediatric Section of the New York Academy of Medicine .. 161, 534, 937, 1147
Rochester Pediatric Society and Central New York Pediatric Club 539
St. Louis Pediatric Society 164, 1146
Spasm, spasmus mutans and nystagmus .. 704
Spasmophilia, cardiac troubles in 325
eggs in diet of spasmophilic patient 326
rachitic 731
Spillman, R.: Studies on hearts of normal children: roentgen-ray studies *791
Spina bifida, lateral; case 531

PAGE

Spine, congenital defects of 674
lesions, use of iodized oil in roentgen-ray diagnosis of 522
splinting operation (Albee's) of vertebrae tuberculosis of, in young children 944
vertebral disease with meningeal symptoms 545
Spleen, effect of spleen marrow extracts on blood regeneration 668
Splenomegaly, familial, two cases 519
lipoid cell splenohepatomegalia in infant. 519
splenomegalic cirrhosis of liver and cholelithiasis occurring in a family 522
Staphylococci, infection of spinal meninges. 1131
Stephens, V. R.: Acute intussusception; manipulative reduction under fluoroscopic control *61
Stewart, C. A.: The Pirquet test; comparison of scarification and puncture methods of application *387
Stoesser, A. V.: The hypertrophies of infancy and childhood *885
Stoloff, E. G.: Acute massive atelectasis of the lung *239
Stomach, cardiospasm 693
cardiospasm in infancy and in childhood; report of case in child 11 years of age *426
congenital nonrotation of, with eventration *640
connection between chlorine content of blood, blood plasma and gastric secretion 673
determination of function of, in infants .. 502
gastric attacks in childhood 320
normal and pathologic physiology of 897
pathogenesis of disturbance of nutrition and digestion 1114
syphilitic tumor of, in girl of 12 years .. 721
scarlatinal, vitality of 498
Streptococci, hemolyticus, presence of, in pharynx and on tonsils in apparently healthy individuals 706
infection mucus, causing lateral sinus thrombosis 152
transmissible toxicogenity of 497
Strophulus, pathology of 529
Strychnine, conditions of toleration to strychnine medication in pediatrics 718
studies in 705
Sugar, treatment, of children with nephritis 1127
Suprarenals, congenital absence of both infrarenal glands 1116
hemorrhage, in case of status lymphaticus 543
neuroblastoma of, in young children 162
tuberculosis of? 547
Sure, B.: Vitamin requirements of nursing young; production of beriberi in nursing young (mus norvegicus albinus) associated with hemorrhages *811
Swanson, W. W.: Congenital cyst of lung *1024
Synthalin, in diabetes mellitus 693
Syphilis: See also Meinicke's Reaction
and injury to the embyro 1124
congenital, bismuth treatment of 681
congenital diagnosis 514
congenital, gummatous renal aplasia 769

Syphilis—Continued PAGE
congenital, neoarsphenamine treatment in infants 515
congenital, of liver 680
congenital, tests for liver function..... 514
congenital, treated with malaria 522
congenital, treatment by intraperitoneal injection of neoarsphenamine: report of 2 years work..... *47
experimental, in rabbit effect of elevation of body temperature on course of..... 313
Froin's syndrome due to..... 736
hereditary, how Vienna is meeting the problem of 1123
hereditary, in foundling..... 142
hereditary, prophylaxis of..... 1122
its social importance in child..... 513
pseudotabes juvenile; case..... 514
syphilitic tumor of stomach in girl of 12 years 721
transmitted by blood transfusion..... 313
treatment in infants 315
Syringomyelia, in childhood..... 704

Tabes Dorsalis, juvenile, three cases.....*866
Tachycardia, paroxysmal, treated with apomorphine 1130
Tannic acid, in treatment of burns....546, 1137
Teeth, factors in preventive orthodontia.... 946
factors of decay in..... 345
milk, congenital syphilitic changes in.... 683
Temperature, high, in infants, due to crying 160
significance of experimental cooling of body with reference to colds..... 128
work (crying) hyperthermia in infants.. 296
Temporal bone, bilateral symmetrical breaking down of..... 1136
Testicle tumors of congenital origin..... 146
undescended 945
Tetanus, following smallpox vaccination... 699
new method of treatment..... 141
Tetany, effect of epinephrine and ergotamine in experimental tetany.....1113
infantile, effect of parathyroid extract on mineral metabolism in.....*392
infantile, sex factor in.....*964
of sphincter of bladder..... 325
spasmophilic diathesis study of..... 526
Thallium acetate, epilation, in treatment of ringworm of scalp.....155, 529
Thorax, roentgenographic changes of, in new born infants, study of 25 cases.*837
Thrombopenia 543
Thrombosis, bilateral of facial and angular veins 550
Thymona, case..... 342
Thymus, clinical manifestations of enlarged 143
crossed dystopia of..... 694
enlargement, as cause of atelectasis.... 145
hypertrophied, in new born.....1147
proliferative reactions of..... 129
spasmophilia with hypoplasia of parathyroids and..... 526
thymic stridor..... 144
Thyroid, hyperthyroidism after feeding fresh thyroid in hypothyroidism..... 520

Thyroid—Continued PAGE
role of thyroid and parathyroid glands in chemical differentiation of bone during growth..... 126
therapy, effect on children.....*203
Tongue, glossitis with leukoplastic patches and facial dermatitis of bacterial origin 156
Tonsils, function, experimental investigation of..... 155
pharyngeal *229
problem of..... 527
question of removing..... 528
Tonsillectomy, early, in peritonsillar abscess 153
Toomey, J. A.: Intradermal vaccination..*186
studies in scarlet fever; skin blanching by commercial antitoxins.....*607
Topper, A.: Effect of thyroid therapy on children *203
Toxin, and antitoxin solutions, phenol and tricresol effects in..... 127
relation of skin sensitivity to, and development of antitoxic immunity during artificial immunization.....*974
Trachoma *294
Trichophytosis, chronic, of skin and nails in all members of a family, with invasion of the mucosae, lymph nodes and bone 333
Tubercle Bacilli, bacterial action of light on 313
Tuberculid, papuloannular..... 681
Tuberculin, immunobiologic (Pirquet) diagnosis of tuberculosis in infancy.... 316
Pirquet test: comparison of scarification and puncture methods of application.*387
reaction, evaluation of..... 681
tests, incidence of tuberculous infection in children from rural districts..... 341
Tuberculosis, allergic manifestations in.. 732
blood picture of latent tuberculosis in children and its prognostic significance 514
case finding in children..... 141
congenital 315
diagnosis of, by culture.....1123
diagnosis, treatment and prevention of 512
differential diagnosis of early pulmonary tuberculosis and mitral disease..... 516
differential diagnosis of tuberculous and non-tuberculous pulmonary disease in childhood 1124
etiology of secondary tuberculous meningitis 1122
in children.....683, 933
in infancy, the Pirquet immunobiologic diagnosis of..... 316
influence of whooping cough on immunity to 682
of abdominal lymph nodes in early life. 511
of spine, in young children..... 944
of urethra 515
pericarditis as primary clinical manifestation of, in childhood.....*631
periomphalitis and perforation through the umbilicus in tuberculous peritonitis.... 682
present status of BCG..... 515
primary complex in children..... 515
prophylaxis of, in infancy..... 512
pulmonary, treatment by artificial pneumothorax 511
radium therapy in tuberculous cervical adenitis 511

Tuberculosis—Continued PAGE
roads of propagation in body of..... 314
sedimentation rate of erythrocytes in infantile 513
social service for children..... 142
surgical, value of sedimentation test in 679
suprarenals? 547
the hilum in..... 311
treatment 680
tuberculous peritonitis, medical or surgical treatment?.....1122
tuberculous ulcer in a nursing infant.. 314
vaccination of new born against..... 681
verrucosa cutis caused by thumb sucking 703
Tularemia, cutaneous manifestations of..1118
Tumors: See also under names of tumors as Endothelioma; Ganglioma; Neuroblastoma Papilloma; Thymona, etc.
cerebellar, occurring in identical twins..1130
intercranial, of childhood.....1143
lymphatic, of intestine, causing obstruction 742
of kidney, surgical results of..... 713
syphilitic, of stomach in girl aged 12 years 721
testicular, of congenital origin..... 146
Twins, hemihypertrophy and twinning.... 133
identification characters of single ovum twins 504
Types, Kretchmer's constitutional types in children of school age.....1140
Typhoid Fever *1073, *1088

Ultraviolet Rays, Bacterial action of light on tubercle bacilli..... 313
direct and indirect light therapy of rickets 904
effect on blood of new-born infants; erythrocytes and hemoglobin..... *9
influence on serum irradiated in vitro.... 668
suggested nomenclature for ultraviolet zones 668
treatment 717
Umbilicas, periomphalitis and perforation through, in tuberculous peritonitis.... 683
Uranin, as aid to diagnosis of meningitis..1134
Urea, clinical value of azotemia in digestive disturbance of infant..... 298
Urethra, tuberculosis of..... 515
Urine, acid-base composition of, from an epileptic child 699
action of urinary antiseptics..... 339
cause of cylinduria in infants..... 520
effect on, from addition of acids and alkalis to the diet of infants..... *38
effects of oliguria on acid base balance of plasma of infants with mastoiditis..*557
enuresis, psychiatric approach to..... 737
influence of albumins on proportion of some nitrogen containing substances in urine 670
serologic study of pyuria of infants.....1129
urobilinuria in children with heart disease 535
urochromogen test in clinical medicine... 670
Urticaria, pigmentosa, case of..... 165

Vaccination, against tuberculosis of new born 681
disturbances of central nervous system... 149
encephalitis after..... 679

Vaccination—Continued PAGE
intracutaneous smallpox 309
intradermal *186
methods of purifying smallpox vaccine.. 297
problem of the "pure" smallpox vaccine in connection with encephalitis postvaccinalis 310
school *1092
Vaccine, antirabies, fatal acute ascending paralysis following use of..... 703
pertussis, potency of stored..... 667
present status of BCG..... 515
Varicella: See Chickenpox
Vein, jugular, pulsation and visibility of, through the intact drum-membrane....1136
Venereal disease.....*1050
Virus, comparison of virus obtained from encephalitis and rabies..... 666
Vital capacity, of the Negro..... 158
Vitamins, decrease of resistance as result of deficiency of vitamin D..... 903
deficiency, specific dynamic action in....1114
relation between skin and..... 671
production of beriberi in the nursing young (mus norvegicus albinus) associated with hemorrhages.....*811
Vomiting, effects of, on acid-base balance of plasma of infants with mastoiditis....*557
persistent, in infants..... 501

Wakeman, A. M.: Mineral metabolism on a high mineral diet.....*576
the effect of parathyroid extract on mineral metabolism in infantile tetany.....*392
Waldo, P. C.: Congenital syphilis; treatment by intraperitoneal injection of neoarsphenamine: report of 2 years work *47
Wang, C. C.: Influence of feeding on certain acids in feces of infants; comparison of effects of breast milk and modified cow's milk on excretion of volatile acids *580
metabolism of undernourished children; calcium metabolism.....*856
metabolism of undernourished children; protein metabolism.....*968
Weight, and height, growth standards of, for girls in private schools.....*411
growth of children below average..... 158
Wetnurses, directory of.....1139
Weymuller, C. A.: Roentgenographic changes in the thorax of normal new born babies; daily roentgenographic study of 25 normal babies during the first fourteen days of life.....*837
Wheelihan, R. Y.: Granulocytic aplasia of bone marrow following use of arsenic *1032
Whooping Cough.....140, *1069
bacteriology of..... 139
development of zoster and varicella following injection of serum..... 156
eclampsia 677
in Sanitoria 507
influence of, on immunity of tuberculosis. 683
microscopic changes in larynx in.....1121
pathologic anatomy of, with special reference to pneumonia caused by the pertussis bacillus 507
prevention and treatment of, by vaccines and ether..... 1120

Whooping Cough—Continued PAGE
sexual differentiation in susceptibility to and mortality from whooping cough in children under 5 years..... 305
treatment of 318
Wile, I. S.: Physicial problems of abnormal behaviour *113
Williams, C. T.: Infrequency of severe rickets in New Orleans and vicinity; an attempt to correlate some of the responsible factors.....*590
Williams, J. L.: Etiology of an epidemic of enteritis associated with mastoiditis in infants *955
Wilson, J. R.: Respiratory metabolism in infancy and in childhood; elimination of water through the skin and respiratory passages of infants..... *54
Williams, J. R.—Continued PAGE
Respiratory metabolism in infancy and in childhood; respiratory exchange in marasmus; basal metabolism.....*615
Wolffian ducts in females, persistence of... 673
Wood, A. A.: Influence of feeding on certain acids in feces of infants; comparison of effects of breast milk and modified cow's milk on excretion of volatile acids *581
Xanthocrania: Froin's syndrome due to syphilis in boy aged 13 years..... 736
Xanthomatosis, cutis, and hypercholesterolemia 332
Xeroderma pigmentosa 161
Zoster, development after injections of serum for whooping cough..... 156

JUN 29 1928

VOLUME 35 NUMBER 6

AMERICAN JOURNAL

OF

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JUNE, 1928

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $5.00

Entered as Second-Class Matter, January 4, 1911, at the Postoffice at Chicago, Illinois, Under Act of Congress of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on June 14, 1918.

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THE AMERICAN JOURNAL OF DISEASES OF CHILDREN is published by the American Medical Association as a means for the advancement of knowledge of the diseases of children and for the publication of scientific investigation in this field.

Manuscripts for publication, books for review, and correspondence relating to the editorial management should be sent to Dr. Clifford G. Grulee, 310 South Michigan Avenue, Chicago, or to any other member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed, AMERICAN JOURNAL OF DISEASES OF CHILDREN, American Medical Association, 535 North Dearborn Street, Chicago.

Articles are accepted for publication on condition that they are contributed solely to the AMERICAN JOURNAL OF DISEASES OF CHILDREN. Manuscripts must be typewritten, preferably double spaced, and the original copy should be submitted. Zinc etchings and halftones of illustrations will be supplied by the Association when the original illustrations warrant.

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THE AMERICAN JOURNAL OF DISEASES OF CHILDREN is published monthly. Annual subscription price (two volumes): Domestic, $5.00; Canadian, $5.40; foreign, $5.75, including postage. Single copies, 75 cents, postpaid.

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CONTENTS

PAGE

The Etiology of an Epidemic of Enteritis Associated with Mastoiditis in Infants. George F. Dick, M.D.; Gladys Henry Dick, M.D., and J. L. Williams, M.D., Evanston, Ill. 955

The Sex Factor in Infantile Tetany. Harry Bakwin, M.D., and Ruth Morris Bakwin, M.D., New York 964

Metabolism of Undernourished Children: V. Protein Metabolism. Chi Che Wang, Ph.D.; Jean E. Hawks, M.S., and Bertha B. Hays, M.S., Chicago 968

Scarlet Fever. Jean V. Cooke, M.D., St. Louis:

VI. The Relation Between the Loss of Skin Sensitivity to Toxin and the Development of Antitoxic Immunity During Artificial Immunization 974

VII. The Course of the Development of Immunity in Scarlet Fever 983

VIII. The Anaphylactic Factor in Scarlet Fever 991

The Hearts of Normal Children: III. Electrocardiographic Records. Edith M. Lincoln, M.D., and Gertrude H. B. Nicolson, M.D., New York 1001

Prevention and Modification of Measles with Antimeasles Diplococcus Goat Serum. Archibald L. Hoyne, M.D., and Silber Peacock, M.D., Chicago 1021

Case Reports:

Congenital Cyst of the Lung. W. W. Swanson, M.D.; E. S. Platou, M.D., and W. Sadler, M.D., Minneapolis. 1024

Granulocytic Aplasia of the Bone Marrow Following the Use of Arsenic. Robert Y. Wheelihan, M.D., Milwaukee 1032

Shoe-Dye Poisoning: Report of Three Cases. John Aikman, M.D., Rochester, N. Y. 1038

Agranulocytic Angina. Hugh L. Dwyer, M.D., and Ferdinand C. Helwig, M.D., Kansas City, Mo. 1041

Progress in Pediatrics:

Survey of the Communicable Disease Situation in Chicago. Jesse R. Gerstley, M.D.; J. C. Geiger, M.D.; I. S. Falk, Ph.D.; I. A. Abt, M.D.; Clifford G. Grulee, M.D., and John F. Norton, Ph.D., Chicago 1048

Pediatric Biographies:

Leonellus Faventinus Victorius; George Khufner, Jr. John Ruhräh, Baltimore 1106

News and Comment 1110

Abstracts from Current Literature 1111

Society Transactions:

Chicago Pediatric Society 1142

St. Louis Pediatric Society 1146

New York Academy of Medicine, Section on Pediatrics. 1147

Northwestern Pediatric Society 1149

Book Reviews 1151

Directory of Pediatric Societies 1153

General Index 1157